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THE
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ORIGINAL COMMUNICATIONS.

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THE EMBRYOLOGY AND NEUROHISTOLOGY OF SPHENOPALATINE GANGLION CONNECTIONS; A CONTRIBUTION TO THE STUDY OF OTALGIA.*‡

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Portland, Ore.

Man's use of sternutatory drugs from the remotest antiquity shows his thought of the nasal mucosa as a portal for influence upon the vital forces and upon the brain. In relatively recent years, the sensory and sympathetic reactions of the nose have tempted various bold theorists to attack distant cranial, visceral or pelvic disorders by cauterization of certain turbinal or septal areas. Fantastic systems of localization were published, with a succession of reputed cures.[1 2 3]

Scientific observation in this field began long after the prompting of Hilton,[4] with the records of neuralgias traced to nasal, sinus and dental disease and studied by Hack,[5] Delavan,[6] Legal,[7] Politzer,[8] Ziem,[9] Ménière[10] and others. Medical measures, and the cautery proving inadequate, clinicians turned to increasingly radical surgery, often with splendid results. Technical difficulties in securing material from the sphenoethmoidal region have, however, delayed investigation of the minute anatomy of Meckel's ganglion and its connections. Most clinicians have been content to rely upon descriptions

*From the Departments of Anatomy and Oto-Laryngology, University of Oregon Medical School, Portland. Read before the American Otological Society, Washington, D. C., May 1, 1928.

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by the earlier microscopists, losing sight of the fact that their own operative technique and therapeutic armamentarium has entirely changed in 30 years.

Foremost in scientific accuracy, in the analysis of otalgia and glossodynia, come the studies of nerve distribution based upon herpes zoster oticus, made between 1907 and 1915 by J. Ramsay Hunt.[11 12 13] Models of completeness, these studies definitely map the extension of sensory filaments through the geniculate ganglion to the auricular region. Though attacked by Mills[14] and Gordon Wilson,[48] this work remains standard and has been brought up to our time by clinical reports from numerous others, notably Emerson,[15] Dennis[16] and Sears.[17] Accurate boundaries for nerve distribution when measured by so violent a ganglionic irritation as that of herpes zoster are of course not always obtainable.

The remarkable generalizations of Sluder,[18] based upon his extensive operative experience and upon his method of destruction of the sphenopalatine ganglion, have since 1900 arrested the attention of clinicians throughout the world. Unquestionably his courage has done much to throw light into one of the darkest borderlands of otolaryngology. One looks in vain, however, even in his latest work, for the detailed anatomic and histologic background found in the work of J. Ramsay Hunt. Reliance upon descriptive anatomists causes Sluder to resort to bold hypotheses; he says, "I have indulged in some speculations which will probably be forever impossible of proof."[19] Although brilliant clinical successes have unquestionably followed his own attacks upon referred pain, there have been disappointments among his followers, and not a few active dissenters among contemporary teachers of equal experience and learning.[20 21]

Sluder's discouragement over our inadequate knowledge of this field is shared by Müller and his collaborators, who remain uncertain as to the function of the deep petrosal,[22] and whether sensory impulses travel along the cervical sympathetic.[23] They regard the study of sympathetic tracts by degeneration methods after section as well-nigh impossible.[24] Halphen comments, "Today the sympathetic is in fashion, and we believe we possess the truth because we attach to the sympathetic what yesterday we attached to anaphylaxis, and day before yesterday to bulbar reflexes. . . . Our pathogenesis appears built upon shifting sands."[25] Bertein states, "We feel the insufficiency of explanations tending to attach these different troubles to sinus infection which is demonstrable neither clinically nor at operation."[26] Terracol considers the sphenopalatine a mixed ganglion, connected with and traversed by mixed nerves; nevertheless he says,

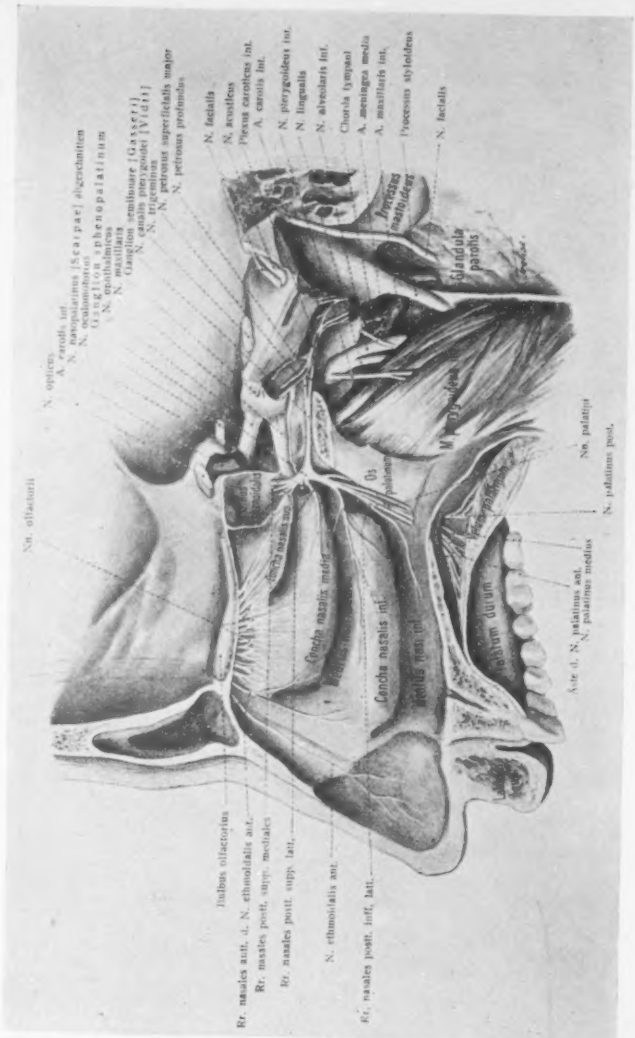

Fig. 1. The sphenopalatine ganglion and its connections viewed from the medical aspect. (From Rauber-Kopsch, Lehrbuch der Anatomie.)

"Its rôle appears formidable, but its pathology remains rudimentary; and it remains really an undiscovered country, perhaps the Promised Land."[27]

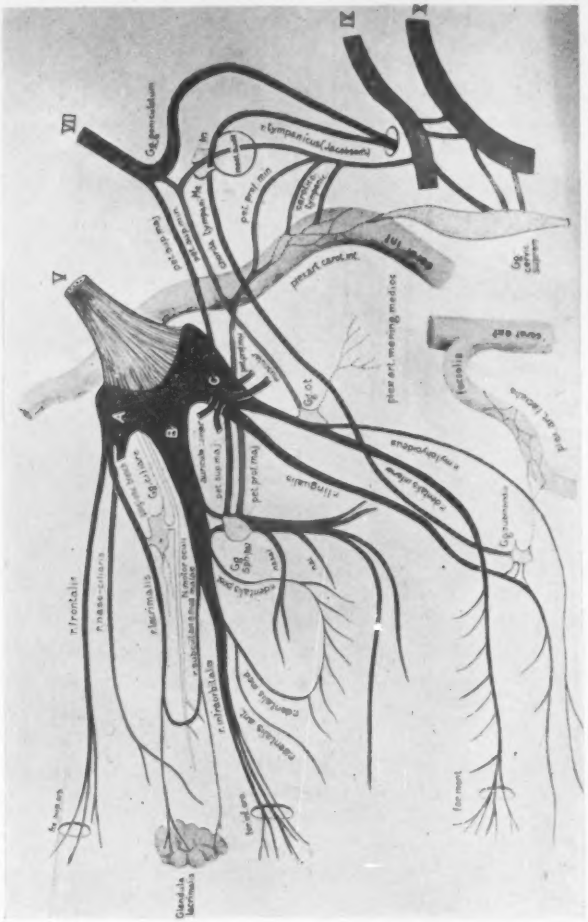

Fig. 2. The interrelation of the Trigeminus, Facialis, Glossopharyngeus and Vagus, together with the sympathetic ganglia in man. (From H. H. Wilder, History of the Human Body; after Gray, Arnold and Gegenbaur.)
Systems: *White*, Motor oculi; *black*, Trigeminus; *dotted*, Sympathetic; *light gray*, Glossopharyngeus and Vagus; *dark gray*, Facialis.

Impelled by these uncertainties among clinicians and teachers, we have felt that the comparative anatomy and embryology of the ganglion and its connections might furnish us with some clues whereby

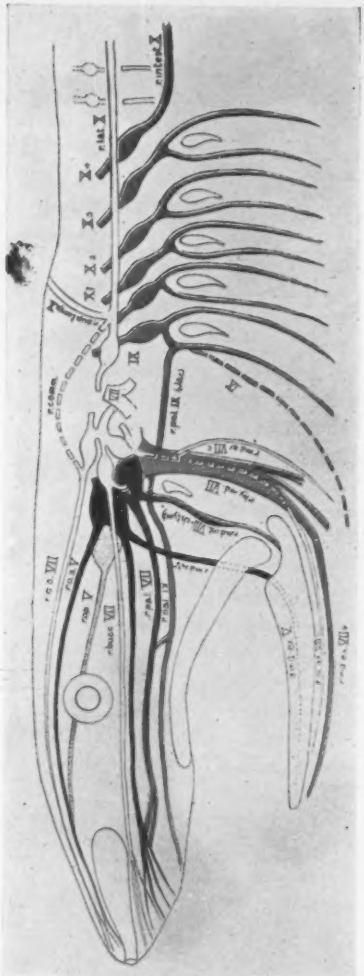

Fig. 3. Diagram of cranial nerves in Anamnia. (From Wilder, after Wiedersheim.) Roman numerals indicate the cranial nerves. Branches of these are designated as follows: *r. o. s.*, ramus ophthalmicus superficialis; *r. o. p.*, ramus ophthalmicus profundus; *r. bucc.*, ramus buccalis; *r. pa.*, ramus palatinus; *r. md. ex.*, ramus mandibularis externus; *r. md. int.*, ramus mandibularis internus; *r. hy. md.*, ramus hyomandibularis; *r. lat.*, ramus lateralis; *r. intes.*, ramus intestinalis. Systems: *white*, entire supply to lateral line organs, also Acusticus; *black*, Trigeminus; *dotted*, ophthalmicus profundus; *dark gray*, Facialis, sensory elements; *light gray*, Facialis, motor elements; *medium gray*, Glossopharyngeus and Vagus.

we might trace the pathways of sensation toward the periphery. Fresh material secured by Dr. Warren E. Hunter, of the Department of Pathology, has permitted modern methods of staining and impregnation. It is hoped that the serial sections and reconstructions thus obtained may assist in solving some of the problems just stated, without reference to debatable theories.

The sphenopalatine ganglion has its embryological origin in mammals by migration of cells from the V and VII ganglia (Kuntz).[28] It is connected, however, in the adult, not only with the maxillary branch of the trigeminus nerve through the sphenopalatine nerves, and with the facialis through the intermediation of the Vidian nerve and the great superficial petrosal nerve, but also with the glossopharyngeus nerve through the great deep petrosal and its continuations, the small deep petrosal and the nerve of Jacobson (Figs. 1 and 2). It is possible that there are also connections with the vagus, through the communicating branches between the nodose ganglion of the latter nerve and the glossopharyngeus nerve.

An understanding of the intricate relations of this centrally located ganglion, with its many connections, must be based upon a knowledge of the comparative anatomy of the region and nerves involved.

In the sharks (Fig. 3), both the facialis and the glossopharyngeus nerves give off a rostrally directed palatine branch. These two rami supply, in general, the same region, and the branches illustrate the fundamental pattern of the cranial nerves as it is conceived by morphologists.[29] No sphenopalatine ganglion as such is present in the sharks. The ramus palatinus VII is derived from the geniculate ganglion, as shown in the figure.

In the amniotes (Fig. 4), conditions are somewhat modified. A ganglion has appeared along the course of the palatine VII, nerve, and the palatine IX nerve has disappeared as a distinct branch. The IX nerve is still connected with the VII nerve through the communicating branch of Jacobson; and in the amniotes we also find a connection, apparently not present in the fishes, in a branch between the ganglion nodosum X and the petrous ganglion of the glossopharyngeal nerve.

The cranial nerves of the lower vertebrates have a somewhat different equipment of functional components from that which persists in the mammals and man. The facial nerve, for example, possesses some five distinct physiological sets of nerve fibres. This is also true of the glossopharyngeal. These components have been thoroughly studied in many fishes and amphibians by Herrick,[30 31] Cole,[32 33] Coghill,[34 35] Norris[36 37] and others. They may be recog-

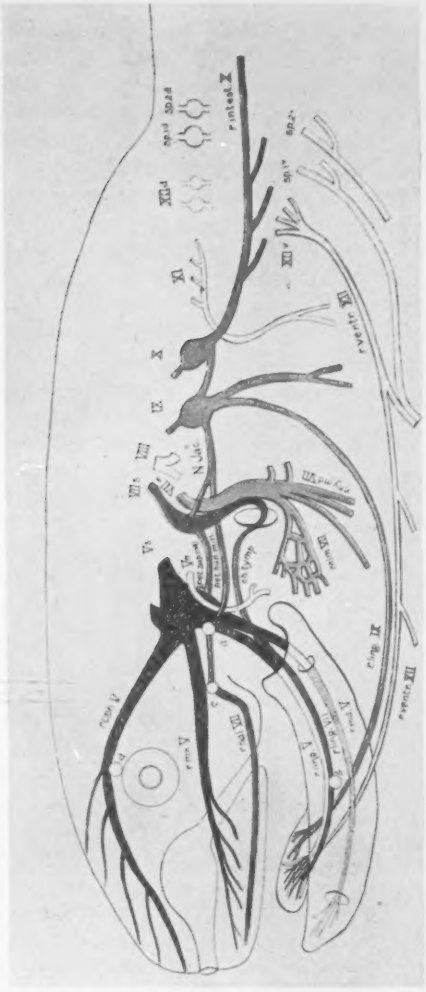

Fig. 4. Diagram of cranial nerves in Amniota (From Wilder, after Wiedersheim.) Roman numerals indicate the cranial nerves. *sp. 1* and *sp. 2* indicate first and second spinal nerves. Branches as follows: *r. oph.*, ramus ophthalmicus; *r. mx.*, ramus maxillaris; *r. pal.*, ramus palatinus; *ch. tymp.*, chorda tympani (ramus mandibularis internus); *r. ling.*, ramus lingualis; *r. md.*, ramus mandibularis; *mim.*, mimetic nerve; *r. hy. md.*, ramus hyomandibularis; *r. ventr.*, ramus ventralis; *pet. sup. maj.*, petrosus superficialis major; *pet. sup. min.*, petrosus superficialis minor; *n. Jac.*, Jacobson's nerve; exponents *d* and *v* denote dorsal and ventral roots; *s* and *m* denote sensory and motor branches; *a*, ciliary ganglion; *b*, submaxillary ganglion; *c*, sphenopalatine ganglion; *d*, otic ganglion. Systems as Fig. 3.

nized by their central connections within the brain, and by their peripheral distribution. There are also certain histological variations among the several types, such as differences in diameter and thickness of myelin sheath, and its presence or absence, differences in the size of fibres, etc., so that the fibres may be followed long distances with assurance. During the process of cephalogenesis and because of the increase in complexity of the central nervous system, components of similar functional type tend to group together, so that within the brain stem we find those of like function from the various cranial nerves entering the same fibre tract; *e. g.*, the visceral afferent fibres of the VII, IX and X all enter the fasciculus solitarius. Similarly the general cutaneous fibres all enter the spinal V tract and nucleus. Peripherally, there is brought about a reapportionment of functional components in the several nerves, so that in mammals, for example, the trigeminus nerve is chiefly sensory and the facialis nerve is chiefly motor, although in the lower vertebrates the two nerves have a very similar equipment of functional components. Particularly has the somatic sensory element of the facialis nerve largely disappeared, while in the trigeminus this component is the most important of all the V nerve system in man. Similar comparisons of other systems can be made, not only in these two nerves, but in others of the cranial series.

With these facts of comparative anatomy in mind, we may now examine more closely the sphenopalatine ganglion and the facialis nerve. The finer structure of the sphenopalatine ganglion has been the subject of many investigations. Retzius[38] (1880) by the method of isolating cells, found in the sheep multipolar cells and in the cat cells with two processes. He stated that in general the ganglion is of the sympathetic type. Müller and Dahl[39] showed by the Bielschowsky technique in 1910 that multipolar cells of the sympathetic type are the characteristic feature of this ganglion (Fig. 5). Müller,[40] in the second edition of his monograph (1924), states that it is made up entirely of multipolar cells, and adds that there can be no doubt of its relation to the sympathetic system. He states that the preganglionic fibres reach Meckel's ganglion from the bulb through the great superficial petrosal nerve. Carpenter,[41] using the methylene blue technique in 1912, demonstrated in the sheep not only the multipolar cells resembling the ordinary type of mammalian sympathetic cells, but he also showed most beautifully the "subcapsular, pericellular end-nets of fine varicose fibrils similar to those of preganglionic fibres in the vertebral and prevertebral ganglia of the sympathetic system" (Fig. 6). These and other studies appear convincing

as to the types of cells present, and their relation to the cranial autonomic system.

Our own observations confirm these statements. In a kitten of two weeks, the head region of which was stained by the pyridin silver method, multipolar cells (Fig. 7 A, C) are clearly shown in Meckel's ganglion. Also pericellular endings of preganglionic fibres from the great superficial petrosal nerve are found. The cells give off delicate potganglionic fibres which leave the ganglion with the

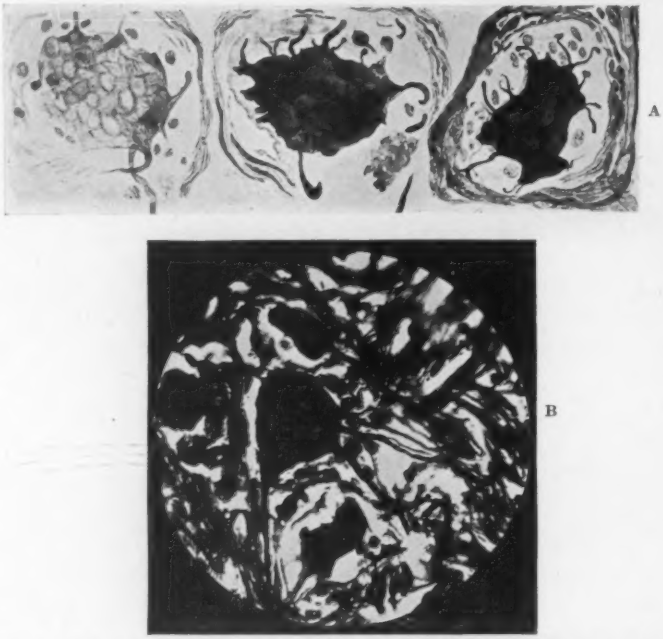

Fig. 5. Cells of the sphenopalatine ganglion, showing multipolar character: A, in man; B, in the horse. (Bielschowsky technique; from Müller and Dahl, Deutsch. Arch. f. klin. Med., 99, 1910.

various nerves that radiate from it. Their ultimate destination we have not followed.

In human material, stained with iron-hematoxylin, osmic acid, and by the Pal-Weigert method, the processes of the nerve cells are not shown. There can, however, be no doubt of the similarity of this ganglion to other sympathetic ganglia, so far as its cells are concerned. Preganglionic fibres, with their myelin sheaths of small

diameter, enter from the great superficial petrosal nerve. There are also larger fibres of the sensory type from this nerve in considerable number. These larger fibres do not terminate in the ganglion, but pass through it into the palatine nerves, and probably into other branches, with which they are presumably distributed to the palate and elsewhere. Bundles of unmyelinated fibres, together with myelinated fibres of the sensory type, also enter from the deep petrosal part of the Vidian nerve, but appear to pass through the ganglion without interruption. This point, however, could not be adequately controlled with the available material. The sphenopalatine branches from the maxillary nerve, in our iron-hematoxylin preparations, ap-

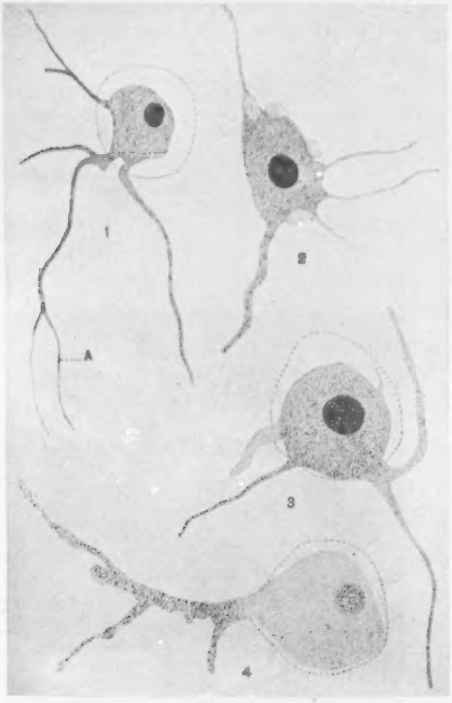

Fig. 6A.

Fig. 6A. A. Cells showing processes, and B. Cells showing pericellular networks of preganglionic fibres, from various cranial autonomic ganglia of the sheep: 1. Cells from submaxillary ganglion; 2. cell from otic ganglion; 3. cell from sphenopalatine ganglion; 4. cell from ciliary ganglion; 5. and 6. end-nets from ciliary ganglion; 7. end-nets from sphenopalatine ganglion, incompletely stained; 8. and 9. end-nets from otic ganglion; 10. end-net from submaxillary ganglion. Methylene blue technique. (From F. W. Carpenter, Jour. Comp. Neur., 22, 1922.)

pear to pass through the ganglion without interruption, continuing into the palatine nerves. This is in general agreement with the observations of Rhinehart[42] in the mouse.

It should be recalled that the facialis is primarily the nerve of the hyoid arch. Its somatic afferent fibres in lower vertebrates reach the general region of the skin which becomes involved in the development of the external ear. The studies of Johnston,[43] Herrick,[30 31] Norris,[36 37] and others, have shown this to be true in fishes and amphibi-

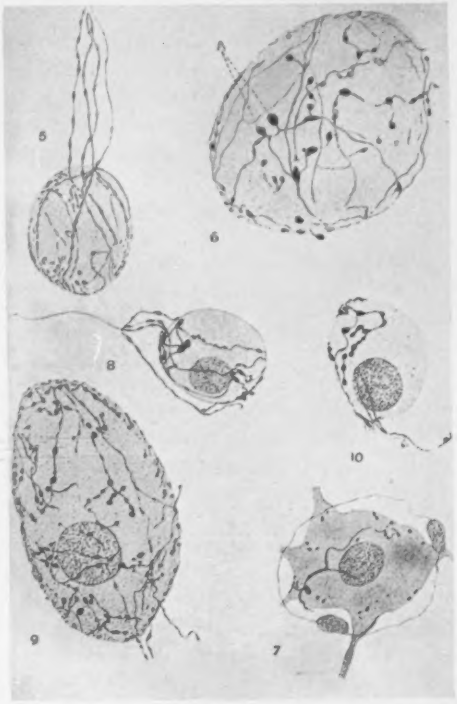

Fig. 6B.

ans. The other components of the facialis are all visceral. They include the general visceral efferent to glands, the special visceral efferent to the hyoid and facial muscles, the general visceral afferent for deep visceral sensibility, and a special visceral afferent to the taste buds on the anterior two-thirds of the tongue, and possibly some on the soft palate (Sheldon[44]).

The special visceral afferent fibres arise from cells in the geniculate ganglion and pass through the chorda tympani and the lingual nerve to the taste buds. This route, long in question, has been strongly supported by the investigations of Cushing[45] by clinical methods, and corroborates the conclusions of comparative anatomists (Sheldon,[44] Herrick[46]). The general visceral afferent fibres are distributed to the deep surfaces through probably all of the facialis branches (Herrick[46]). The other component of the facialis to which reference has been made and which demands our special attention is the general somatic afferent. This component has also been demonstrated anatomically in the mouse, in which it passes to the skin of the back of the ear (Rhinehart[42]), after anastomosing with the ramus auricularis vagi. In addition it supplies part of the external auditory meatus and the tympanic membrane (Rhinehart). Aside from these VII fibres, the nerve supply of the tympanum according to Rhinehart it as given by J. Gordon Wilson.[47 48] This ramus cutaneus facialis of Rhinehart has not been demonstrated anatomically in man, but J. Ramsay Hunt[11 12 13] has brought forth very considerable evidence of its presence by clinical studies. A number of textbooks of anatomy describe and figure an anastomosis between the facialis and the auricular branch of the vagus corresponding to that described by Rhinehart (Quain, Toldt, Piersol and others).

We have found a small branch, in a human fetus of 54 m.m. crown-rump length, which corresponds very closely to that described by Rhinehart in the mouse. In our serial sections, stained with hematoxylin and eosin, we were not able to differentiate size of fibres, but the branch in question anastomoses with the auricular branch of the vagus; and presumably the facial fibres are distributed as in the mouse, namely, through the ramus auricularis vagi to supply a part of the external auditory meatus, the tympanic membrane, and the skin of the cranial side of the concha. In our series this branch is given off close to the chorda tympani but a little distal to the latter. We have also found another branch of the facialis more distally which may correspond to the cutaneous filaments of the facial nerve to the auricle described by Valentin[49] in the adult human. This branch in the embryo is directed toward the auricle, but it was not possible to trace it to the surface with certainty.

The palatine branch of the facial, described by Herrick and other authors as visceral sensory in lower vertebrates, has its morphological equivalent in man in the great superficial petrosal nerve. In the forms above the fishes, in which the sphenopalatine ganglion is represented along the course of this nerve, another element enters; namely,

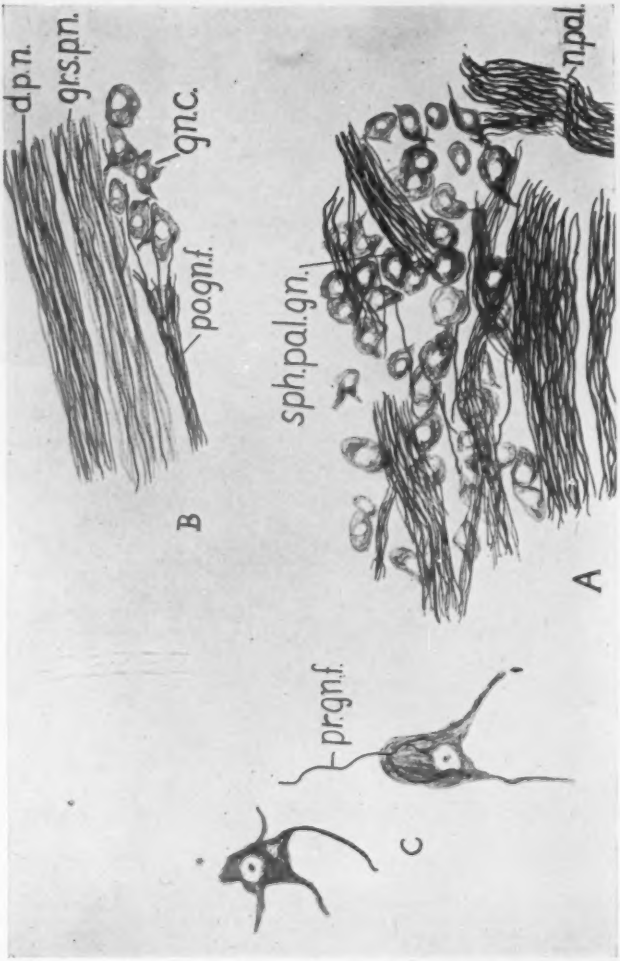

Fig. 7. A. Portion of the sphenopalatine ganglion. B. Portion of Vidian nerve, with small cluster of ganglion cells. C. Sphenopalatine cells, showing multipolar type and portion of an end-net of a preganglionic fibre. *d. p. n.*, deep petrosal nerve; *gn. c.*, ganglion cells; *gr. s. p. n.*, great superficial petrosal nerve; *n. pal.*, palatine nerve; *po. gn. f.*, postganglionic fibres, having their origin from the ganglion cells; *pr. gn. f.*, preganglionic fibre terminating in end-net about ganglion cell; *sph. pal. gn.*, sphenopalatine ganglion. Kitten of two weeks. Pyridin-silver technique. Camera lucida drawings. A. and B., X470; C., X960. (Larsell.)

preganglionic fibres to this ganglion. The great superficial petrosal on this basis may be considered a mixed trunk composed of the equivalent of a white ramus communicans and the palatine VII. His-

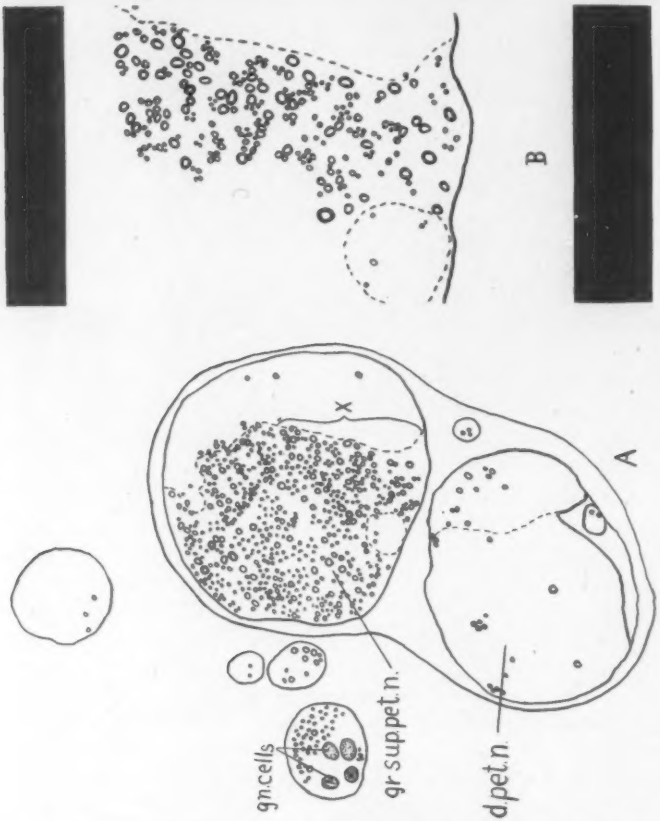

Fig. 8. A. Section of the nerve of the Vidian canal showing myelinated nerve fibres. The fibres of large diameter are of sensory type, those of small diameter are preganglionic fibres to the sphenopalatine ganglion.

B. Portion of A, indicated by X, under higher magnification. Human material fixed and stained in osmic acid within 24 hours after death. *gn. cells*, ganglion cells; *gr. sup. pet.*, great superficial petrosal nerve; *d. pet. n.*, deep petrosal nerve.

Fig. A. drawn with aid of Edinger-Leitz projection apparatus; Fig. B. drawn with camera lucida. (Larsell.)

tological studies of mammalian material confirm this interpretation. Rhinehart[42] found in silver preparations that many of its fibres arise, in the mouse, from cells of the geniculate ganglion. Van Gehuchten[50]

found that some cells of the geniculate ganglion degenerate after section of the facial nerve at its exit from the facial canal. Yagita[51]

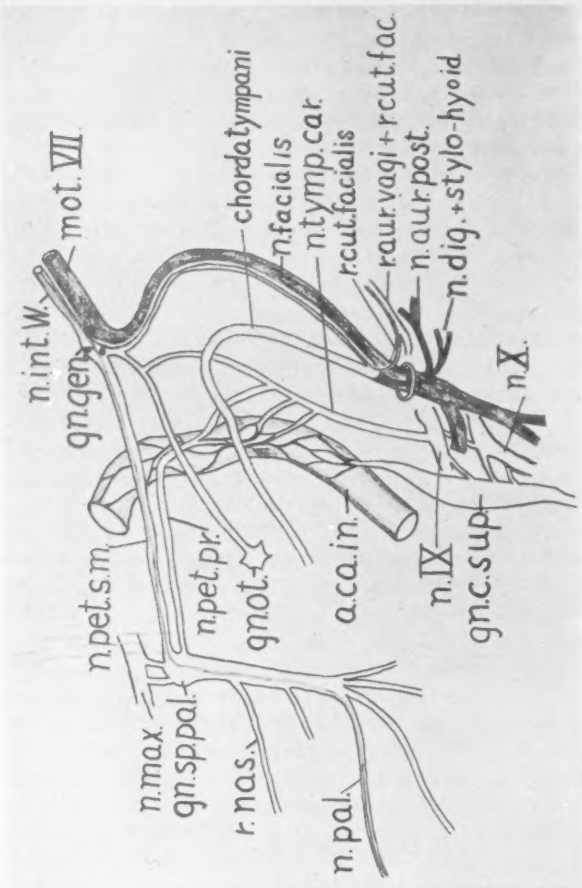

Fig. 9. Diagram of sphenopalatine ganglion and its connections, showing probable pathway of reference of pain in otalgia from involvement of sphenopalatine region. Motor portion of facialis shaded. *a. ca. in.*, internal carotid artery; *gn. c. sup.*, superior cervical ganglion; *gn. ot.*, otic ganglion; *mot. VII*, motor root of facialis nerve; *n. aur. post.*, posterior auricular nerve; *n. dig. + stylohyoid*, nerve to posterior belly of digastric and to stylohyoid muscles; *n. int., W.*, intermediate nerve of Wrisberg; *n. max.*, maxillary nerve; *n. pal.*, palatine nerve; *n. pet. pr.*, deep petrosal nerve; *n. pet. s. m.*, great superficial petrosal nerve; *n. tymp. car.*, carotico-tympanic nerve; *n. nas.*, nasal nerve; *n.X*, vagus nerve; *n.IX*, glossopharyngeal nerve. (Larsell.)

found that about one-twelfth of the cells of the geniculate ganglion showed chromatolysis following section of the great superficial petrosal.

In spite of much confusion in textbooks and the literature regarding the geniculate ganglion, it appears that its cells are of the unipolar sensory type (Retzius,[38] Lenhossék,[52] Müller and Dahl,[39] Müller,[40] Rhinehart[42] and others), and that it belongs to the craniospinal series of sensory ganglia. Sections of the great superficial petrosal nerve (Fig. 8) show that it contains large myelinated fibres of the sensory type. Presumably it was cutting of these fibres which produced chromatolysis of the ganglion cells in Yagita's experiment. Müller and Dahl also found in this nerve myelinated fibres which they regard as sensory. In addition to the large myelinated fibres, there are numerous small fibres with myelin sheaths, 2 to 4 microns in diameter. These correspond to preganglionic autonomic fibres elsewhere in the body.

In our pyridin-silver series of a kitten of two weeks, there are present also unmyelinated fibres, both of small size and of larger diameter. The small fibres represent postganglionic fibres from scattered groups of ganglion cells along the course of the nerve (Fig. 7B), but this is not true of the larger fibres. It is of course possible in so young an animal that they represent sensory fibres which have not yet acquired a myelin sheath. Rhinehart's observations on the mouse led him to conclude that "both the chorda tympani and the great superficial petrosal nerve contain afferent fibres whose cell bodies are located in the geniculate ganglion, and efferent fibres which pass through the ganglion without connection with its cells."[42] This is in agreement with our conclusions based on human material.

General visceral afferent fibres are probably distributed through most of the branches of the facialis (Herrick[46]), and therefore also by those rami which pass through the sphenopalatine ganglion. Large myelinated fibres may be followed in our series from the palatine nerves, through the sphenopalatine ganglion without interruption, and into the great superficial petrosal nerve. Presumably these are connected with cells in the geniculate ganglion, and represent the fibres cut by Yagita, which produces degeneration of cells in this ganglion.

In the geniculate ganglion are found also the cells of Rhinehart's cutaneous auricular branch of the facialis. We have here the conditions found elsewhere in the body for reference of pain from a visceral region to a body surface, in that the ganglion cells of afferent fibres of both are located in proximity in the same sensory ganglion, and centripetal processes of both sets of ganglion cells pass together into the bulb (Fig. 9). Irritation due to infection, pressure or infiltration in the region of the sphenopalatine ganglion would stimulate the palatine VII and other visceral sensory fibres. These, passing through the great superficial petrosal nerve to the geniculate ganglion,

would come into relation with the somatic sensory cells and fibres of the ramus cutaneus facialis, which has its perpheral distribution to the skin of the lower posterior part of the auricle and over the mastoid process.

It is not necessary to discuss in this connection the mechanism of referred pain. This is a complex problem, whose solution, from the anatomical side, is far from having been accomplished. If one accepts the modified conception based on the histological studies of Dogiel,[53] now held by many, that reference of pain from visceral organs to body surfaces is due to transference of the impulse from the afferent visceral fibres to somatic ganglion cells in the dorsal root ganglion, or its equivalent, the relation of the ramus cutaneus facialis to the geniculate ganglion furnishes the necessary anatomical conditions. If, on the other hand, reference of pain should prove to be due to anatomical or physiological relations of visceral and somatic afferent fibres within the central nervous system, the conditions for this would also seem to obtain in the connections of the nerves under consideration within the medulla oblongata.

Further work is needed along various lines opened up in this study. Connections of myelinated, perhaps sensory fibres traveling in the carotid plexus must be traced. These sensory fibres, coming down from the deep petrosal nerve, appear to be shunted around the carotid plexus into connection with the glossopharyngeus and through it with the vagus. Extensive work in embryonal and adult tissues will be required to clarify these points, and our studies in this region are as yet but fragmentary.

Questions relative to the physiological activities of the sympathetic connections of the sphenopalatine ganglion are not a part of this study. It is possible, however, that changes in sympathetic tonus, and thus in vasomotor conditions, in the crowded nasal, dental and pharyngeal area, may account for some of the pain impulses whose paths we have discussed.[54 55 56 57] Such cases, wherein no local inflammatory reactions are evident, may be conditioned by disturbances in the sympathetic at a considerable distance.[58 59 60] Surgery of the sympathetic, at least so far as the superior cervical ganglion and carotid plexus are concerned, has up to the present not received much support because of annoying ophthalmic symptoms.[61 62] Rufus Cole has recently stated . . . "if scientists have a dogma, it is that the advancement of knowledge can best be accomplished by pursuing it quite independently of any immediate results."[63] Following this precept, we should examine further into the details of these "nerves of life", before advocating radical measures which attempt relief of pain through empirical hypotheses.

BIBLIOGRAPHY.

1. Bonnier, P.: L'Action Directe sur les Centres Nerveux. Paris, pp. 199-200; 1913.
2. Fliess, W.: Neue Beiträge zur Klinik u. Therapie der Nasalen Reflexneurosen. Vienna, 1893.
3. Byrd, H.: Pathological Impulses or Currents. *Med. Jour. and Record*, 121:141-144, 1925.
4. Hilton, John: On Rest and Pain. 2nd N. Y. ed., 1879, pp. 40-42, 1860.
5. Hack, W.: Reflexneurosen und Nasenleiden. Berl. klin. Wochensch., 19:379-384, 1882.
6. Delavan, D. Bryson: Otalgia from Reflex Dental Irritation. *Amer. Jour. Otol.*, 4:177, 1882.
7. Legal, E.: Ueber eine öftere Ursache des Schläfen und Hinterhauptskopfschmerzes. (Cephalalgia pharyngo-tympanica.) Deutsch. Arch. f. klin. Med. Leipz., 40:201-216, 1886-7.
8. Politzer, A.: Lehrbuch der Ohrenheilkunde. Vienna, p. 490, 1882.
9. Ziem, C.: Ueber die Beziehungen zwischen Nasen und Ohrenkrankheiten. Monatsschrift f. Ohrenheilk., p. 371, 1885.
10. Meniere, E.: Cephalée quotidienne guerie par cautèrization nasale et ablation des adénoides. Rev. mens. de lar., 8:401-405, 1888.
11. Hunt, J. Ramsay: Herpetic Inflammations of the Geniculate Ganglion; A New Syndrome and Its Complications. *Jour. Nerv. Ment. Dis.*, 34:73, 1907.
12. Hunt, J. Ramsay: A Further Contribution to the Herpetic Inflammations of the Geniculate Ganglion. *Amer. Jour. Med. Sci.*, 136:226, 1908.
13. Hunt, J. Ramsay: The Sensory Field of the Facial Nerve, etc. *Brain*, 38:418, 1915.
14. Mills, Chas. K.: The Sensory Functions Attributed to the Seventh Nerve. *Jour. Nerv. Ment. Dis.*, 37:273, 1910.
15. Emerson, F. P.: Two Cases of Herpes Zoster Oticus. The Laryngoscope, 34:137, 1924.
16. Dennis, F. L.: Herpes Zoster Oticus. The Laryngoscope, 35: 665, 1925.
17. Sears, W. H.: Herpes Zoster Oticus. Tr. Amer. Acad. O. O., pp. 406-431, 1926.
18. Sluder, G.: Nasal Neurology, Headaches and Eye Disorders (bibl.). St. Louis, 1927.
19. Sluder, G.: Loc. cit., p. 100.
20. Casselberry, W. E.: Attenuated Types of Suppurative Sphenoiditis. Tr. Amer. Lar. Asso., p. 99, 1911.
21. Shambaugh, George E.: Recent Problems in Rhinology. *Minn. Med. Jour.*, 7:496-500, 1926.
22. Muller, L. R.: Die Lebensnerven. 2nd ed., Berlin, p. 130, 1924.
23. Buscher, J. in Müller. Loc. cit., p. 560.
24. Buscher, J. in Müller. Loc. cit., pp. 569-70, 574.
25. Halphen, E.: Les phénomènes sympathiques en rhinologie. Arch. Int. Lar., 5:267, 1926.
26. Bertein, P.: Le système sympathique de la muqueuse pituitaire. Arch. Int. Lar., 4:520, 1925.
27. Terracol, J.: Le ganglionsphéno-palatin. Arch. Int. Lar., 4:818, 1925.
28. Kuntz, A.: Development of Cranial Sympathetic Ganglia in the Pig. *Jour. Comp. Neur.*, 23:71, 1913.
29. Wilder, H. H.: History of the Human Body. 2nd ed., New York, 1923.
30. Herrick, C. J.: The Cranial and First Spinal Nerves of Menidia; A Contribution Upon the Nerve Components of the Bony Fishes. *Jour. Comp. Neur.*, 9:153, 1899.
31. Herrick, C. J.: A Contribution Upon the Cranial Nerves of the Codfish. *Jour. Comp. Neur.*, 10:265, 1900.
32. Cole, F. J.: On the Cranial Nerves of Chimera, etc. Discussion of the Lateral Line system, and the Morphology of the Chorda Tympani. Trans. Roy. Soc. Edin., 38:228, 1896.
33. Cole, F. J.: Structure and Morphology of the Cranial Nerves and Lateral Sense Organs of Fishes, etc. Trans. Linn. Soc. Zool. Sec., 7:115, 1898.

34. COGHILL, G. E.: The Cranial Nerves of Amblystoma Tigrinum. *Jour. Comp. Neur.*, 12:205, 1902.
35. COGHILL, G. E.: Cranial Nerves of Triton Taeniatus. *Jour. Comp. Neur.*, 16:247, 1906.
36. NORRIS, H. W.: Cranial Nerves of Amphiuma Means. *Jour. Comp. Neur.*, 18:527, 1908.
37. NORRIS, H. W.: Cranial Nerves of Siren Lacertina. *Jour. Morph.*, 24:285, 1913.
38. RETZIUS, G.: Untersuchungen über die Nervenzellen der cerebrospinalen Ganglien und den übrigen peripherischen Kopfganglien mit besonderer Rücksicht auf die Zellenauslaufer. Arch. f. Anat. u. Physiol., Anat., abt. p. 369, 1880.
39. MULLER, L. R., and DAHL, W.: Die Betileigung des sympathischen Nervensystems an der Kopfinnervation. Deutsch. Arch. f. klin. Med., 99:48, 1910.
40. MULLER, L. R.: Die Lebensnerven. 2nd ed., Berlin, p. 614, 1924.
41. CARPENTER, F. W.: Histology of the Cranial Autonomic Ganglia of the Sheep. *Jour. Comp. Neur.*, 22:447, 1912.
42. RHINEHART, S. A.: The Nervus Facialis of the Albino Mouse. *Jour. Comp. Neur.*, 30:81, 1918.
43. JOHNSTON, J. B.: The Cranial Nerve Components of Petromyzon. Morph. Jahrb., 34:149, 1905.
44. SHELDON, R. E.: The Phylogeny of the Facial Nerve and the Chorda Tympani. Anat. Rec., 3:593, 1909.
45. CUSHING, HARVEY: The Taste Buds and Their Independence of the Nervus Trigeminus. J. Hopkins Hosp. Bull., 14:71, 1903.
46. HERRICK, C. J.: Introduction to Neurology. 4th ed., Philadelphia, 1927.
47. WILSON, J. G.: The Nerves and Nerve Endings in the Membrana Tympani of Man. *Jour. Comp. Neur.*, 17:459, 1907.
48. WILSON, J. G.: The Nerves and Nerve Endings in the Membrana Tympani of Man. *Amer. Jour. Anat.*, 11:101, 1910-11.
49. VALENTIN, in Testut: Traité d'Anatomie Humaine. 3:728.
50. GEHUCHTEN, A. VAN: Recherches sur la termination centrale des nerfs sensibles peripheriques. 1. Le nerf intermédiaire de Wrisberg. Le Nevraxe, 1:3, 1900.
51. YAGITA, K.: Einige Experimente an dem Nervus petrosus superficialis major zur Bestimmung des Ursprungsgebietes des Nerven. Folia Neurobiologica, 8:361, 1914.
52. LENHOSSEK, M. VON: Das Ganglion geniculi nervi facialis und seine Verbindungen. Beiträge zur Histologie des Nervensystems und der Sinnesorgane. Wiebaden, 1894.
53. DOGIEL, A. S.: Der Bau der Spinalganglien des Menschen und der Säugetiere. Jena, 1908.
54. RAMADIER, J.: Sinusites posterieures latentes; origine de nevrites optique, trigemellaire et sphénopalatine. Ann. d. Mal. de l'Oreille, etc., 41:150-184, 1922.
55. RAMADIER, J.: Topographie des troubles sensitifs au cours des affections sphénoethmoidales posterieures. Rev. d'Otol-neuro-ocul., 1:321-325, 1923.
56. HUBERT, L.: Mechanism of Pain As Seen in Otological Cases. THE LARYNGOSCOPE, 33:596-608, 1923.
57. WORMS, G.: Cephalées et troubles oculaires d'origine sinuso-nasale. Rev. d'Oto-neuro-ocul., 2:494-512, 1924.
58. MULLER, L. R.: Loc. cit., p. 77, 1924.
59. SENSENEY, E. T.: Relation of Endocrine Changes to Sphenopalatine Ganglion Neurosis. Ann. O., R., L., 34:183, 1925.
60. HALPHEN, E.: Les phénomènes sympathiques en rhinologie. Arch. Int. Lar., 5:406-443, 1926.
61. RANSON, S. W.: Anatomy of the Sympathetic Nervous System With Respect to Sympathectomy. *Jour. Amer. Med. Asso.*, 86:1886, 1926.
62. CASTERAN, E.: La sympathicectomie périartérielle comme traitement de l'ozène. Arch. Int. Lar., 6:810-815, 1927.
63. COLE, RUFUS: Interrelation of the Medical Sciences. Science, 67:48, 1928.

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FOREIGN BODY IN NOSE, DIAGNOSED AS CARCINOMA, UNEXPECTEDLY REMOVED.*‡

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This is the case of a girl, age 16 years, whose illness so far as can be ascertained began Dec. 27, 1926. Her previous personal history was not significant except that she had been suffering with headache for the past seven years and that she had been without her sense of smell for five months. Her illness began with a chill that lasted a few minutes. In addition she had a severe headache. That same day she had a flushed, painful face and complained of a general weakness.

At this time there was a swelling over the left malar region which also involved both lids of the left eye. Her condition was diagnosed as erysipelas. At the end of two weeks her erysipelas had cleared up but it was noted that there was an abscess draining profusely through a small opening in the region of the inner canthus of the left eye. She was shortly after seen by the ophthalmologists, who treated her local eye condition. On Jan. 12, 1927, an incision was made into the swollen area near the inner canthus but very little pus was encountered.

Her local condition continued unchanged until March 15, when I was asked to see her. At this time there was a draining sinus in the naso-orbital region, and she complained of left frontal headache. A diagnosis of perforation of the ethmoidal labyrinth was made. The nasal examination revealed a left nasal chamber filled with an irregularly granular mass which obstructed the nasal passage. This mass bled profusely when touched with a probe. It was thought that we were dealing with a neoplasm of unusual vascularity. The Roentgen report of Dr. R. A. Rendich was as follows: "There is a marked decrease of illumination of the left ethmoid sinus area extending into the naris. There is also involvement of the maxillary sinus of this side. The findings suggest a new growth."

Six days later the patient was taken to the operating room, where it was our purpose to perform an external operation of the Moure type, for the radical removal of the new growth. When the patient

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‡Department of Laryngology, Kings County Hospital, Service of Dr. H. Arrowsmith.
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had been anesthetized, the extent of the involvement and the bleeding, which was quite profuse at the slightest contact of the probe, led us to abandon the operation.

It was decided to remove a specimen and then to determine whether operation at a future date would be advisable. In attempting to remove a specimen with a pair of punch forceps, the instrument seemed to remain fast in the tissues, and could not be dislodged. In trying to disengage the forceps, a sudden lack of resistance was felt, and the forceps were removed attached to an irregular, hard,

Fig. 1. Roentgen film showing extensive and irregular shadow in left side of nose, suggesting malignancy.

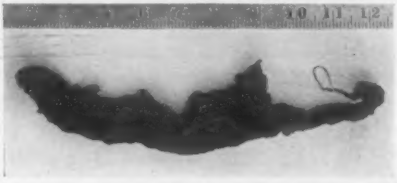

Fig. 2. Piece of gauze removed from nasal chamber. The gauze has formed a cast of part of the nasal passage.

inflexible body, impregnated with blood and tissue juices, and covered with granulation tissue. It was 4½ inches long. Upon examination this proved to be a piece of gauze. The nose was packed with iodoform gauze and a postnasal tampon was used for the control of hemorrhage, which was profuse. The bleeding prevented an examination of the nasal structures to determine the extent of the damage which had been caused by the lodgement of the gauze.

We were unable to obtain a history of any experience which might have a bearing upon the nasal condition from the patient or members

of her family. The patient did not recall ever having had a nasal operation or ever having had her nose packed for bleeding. Repeated inquiries were unproductive of information.

Five days after the removal of the foreign body, examination of her nose showed granulations over the entire lateral wall. The middle and inferior turbinate bodies were flattened against the lateral wall. The septal mucosa of the left side was irregular and healing granulations were to be seen. The external wound stopped discharging and had completely healed ten days after the removal of the foreign body.

The Roentgen report of a film taken nine days after the removal of the gauze stated that the illumination of the left ethmoid and maxillary areas was more marked than when previously examined.

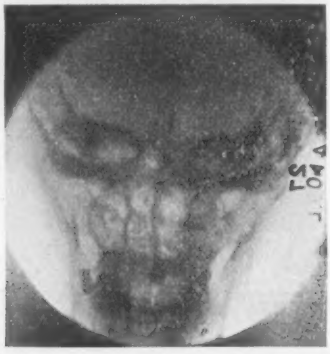

Fig. 3. Roentgen film taken nine days after removal of the foreign body. Note increased clearness of left side of nose as compared with Fig. 1.

The patient was discharged well 13 days after the removal of the foreign body. At this time, as well as since the removal of the gauze, she was free from head ache for the first time in seven years. Her sense of smell at this time was not completely restored.

In this case a large piece of gauze was introduced into the nose which, after the initial reaction following its introduction, remained there by virtue of the tolerance of the nasal structures for it. At no time during the three months while this patient was observed was there any discharge from the nose, the orbital opening evidently being ample to carry off the exudate as it was formed. There was no history which might help in a better understanding of the case either before or after removal of the foreign body.

30 E. 40th Street.

RHINOLITH, REQUIRING EXTERNAL NASAL OPERATION FOR ITS REMOVAL.*‡

DR. M. C. MYERSON, New York City.

In this case the foreign body originated within the nasal passages of the individual; the foreign body was a rhinolith. In the days when people's noses were not as frequently or as routinely examined as they are today, large rhinoliths were not as rare of occurrence. Today they are recognized early while still small. Most of the following has been extracted from the textbook of Sir St. Clair Thomson.[1] More than 300 rhinoliths have been recorded. In neglected cases they attain a large size, as in the case of Melay, where the stone weighed 85 gm. and caused symptoms which simulated a malignant growth. The largest rhinolith recorded was as large as a hen's egg. It weighed 110 gm. and had destroyed the septum and the turbinates. A Rouge operation (sublabial rhinotomy) was necessary for its removal. This was the case of Botey. The surface of the rhinolith is rough, and the stone itself is brittle. The color is reddish-gray, or a darker color of brown or black.

Case Report: M. McD., male, age 69 years, was admitted to the Nose and Throat Service, Jan. 24, 1927, complaining of obstruction to the right side of his nose. He also complained of excessive tearing from the right eye. He could recall that he had had trouble with the right side of his nose for the past four years. During this time this side of his nose gave forth a discharge. About three years ago a foul odor developed. The obstruction gradually increased until it became complete about two years ago. At one time a large mass of putty-like substance was expelled. The patient suffered with frequent colds.

Examination revealed an evident swelling of the soft tissues of the right side of the nose; the cheek partook somewhat of this prominence. A polyp hung down and obstructed the view of the right side of the nose. A thick grayish discharge came from around the polyp and had a foul odor. The left side of the nose was obstructed by a displacement of the septum toward that side. X-ray study by Dr. R. A. Rendich revealed involvement of the right frontal, ethmoid and maxillary sinuses, also the left maxillary sinus. In the right side

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of the nose there was an irregular body which was either a rhinolith or a bony mass; more probably the former.

Attempts to grasp the calculus and remove it were unsuccessful, as were also the attempts to break up the stone. An external operation for delivery of the stone was therefore found necessary.

Under novocain infiltration anesthesia an incision was made along the nasobuccal groove from the naso-orbital region down to the lateral alar attachment at the nasolabial juncture. The tissues were cut

Fig. 1. Roentgen film of lateral view of nose showing rhinolith in situ.

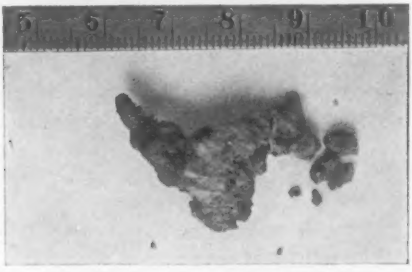

Fig. 2. Rhinolith, weighing 56 gm.

through and the right nasal chamber exposed. A large, irregular stone was found firmly wedged between the septum and the lateral wall. The stone at the time of removal was 4 c.m. by 2½ c.m. in its greatest diameters. The weight was 56 gm. Considerable cheesey matter was encountered filling the nasal cavity. This was removed by suction. The nose was cleansed as much as possible and packed with iodoform gauze to control bleeding. The external wound was

closed with horse hair sutures. A fragment of the stone, together with some of the cheesey matter and a specimen of tissue, were sent to the laboratory. The cheesey substance was reported as necrotic material; the tissue showed chronic inflammatory changes. The stone was analyzed chemically. The stone consisted mainly of calcium carbonate, calcium phosphate and calcium oxalate. The central portion of the stone consisted of organic matter.

One week later examination revealed a synechia of loose macerated tissue extending across the nasal cavity. What was formerly the inferior turbinate was now a mass of soft chronic inflammatory tissue, loosely attached. The nasal cavity again contained considerable of the cheesey substance which was noted at the time of operation. This was entirely removed by suction. The mucosa of the septum on the right side was absent and the quadrilateral cartilage in its central portion was missing. The septal mucosa of the left side was apparently intact; the left chamber was completely obstructed by the displacement of the septum.

The patient was discharged as recovered 12 days after operation. The wound had healed nicely and line of incision was not noticeable.

BIBLIOGRAPHY.

1. THOMSON, SIR ST. CLAIR: Diseases of the Nose and Throat. D. Appleton & Co., N. Y., p. 189.

30 East 40th Street.

AGRANULOCYTIC ANGINA, REPORT OF TWO CASES.

DR. J. J. THOMSON, Mount Vernon, N. Y.

The following cases of agranulocytic angina are reported for the purpose of having them included in the literature of this comparatively rare condition, and because they are fairly typical of the pharyngeal type associated with the unusual blood picture.

Case 1: Miss L. S., age 34 years. Patient was seen in consultation with Dr. Oliver Austin, of Tuckahoe, to whom I am indebted for the early history. Childhood uneventful. During late twenties patient developed depressive maniac psychosis, necessitating institutional care for 18 months. During this time, there was sugar in the urine, increased blood sugar and recurrent attacks of secondary anemia.

Dec. 14, 1927: Dr. Austin found the mental condition more disturbed than usual, and a secondary anemia was present. Under

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treatment there was improvement until Feb. 20, 1928, when the patient complained of a small ulcer on the mucous surface of the upper lip in the median line. This was covered with a gray exudate, tongue coated, marked constipation.

Feb. 22, 1928: The labial ulcer had doubled in size, and the mental condition was much disturbed.

Feb. 23: A new area of ulceration appeared on the right lateral border of the tongue posteriorly. Smears from these ulcers were negative for Vincent's spirilla and fusiform bacilli on this date.

Feb. 24: Area of ulceration increased in size and smear showed bacilli resembling fusiform bacilli and some spirochetes. Culture showed staphlococcus aureus and streptococci. Suspension of neosalvarsan 20 per cent in glycerin was used locally.

Feb. 28: Considerable increase in the size and depth of ulceration on lip.

March 1: Small area of ulceration appeared on right tonsil. Neosalvarsan given intravenously.

March 4: All areas of ulceration have increased; patient is apathetic; temperature 104°.

March 6: Seen in consultation with Dr. Austin. Patient was difficult to handle on account of her general apathy and depressed mental condition. Large burrowing ulcers on upper lip; also on tongue, right tonsil and anterior pillar of right tonsil. Infection had extended to the gums and tooth roots on the upper maxilla. Smears still showed fusiform bacilli and spirilla. Sloughs were removed and neosalvarsan 20 per cent in glycerin used locally. Temperature 104°.

March 8: Ulceration has extended to most of the teeth on the upper maxilla, and a new area has developed over the hard palate on the left side about 3 c.m. in diameter. The slough was removed and neosalvarsan suspension applied to the denuded area. The patient's general condition was not good. Temperature remains high, 104°.

Blood examination showed: red count, 3,900,000; hemoglobin, 72 per cent; leucocytes, 1,000; polymophonuclear, 0; lymphocytes, 100 per cent; no other cells found. Smear taken shows no fusiform bacilli or spirilla.

March 9: *Differential white count showed:* leucocytes, 1,100; polymorphonuclear, 0; lymphocytes, 100 per cent. Transfusion, 500 c.c. whole blood given. Patient expired at 8 p. m.

Case 2: Mr. W. H. L., age 58 years. Occurred in the practice of Dr. Walter Brundage, Pelham, N. Y., to whom I am indebted for the following history.

Aug. 1, 1924: Complained of slight soreness in his throat and slight chilly sensations.

Aug. 2: His past history and family history were both essentially negative. Physical examination showed a well developed and well nourished man. There was a marked pallor of the skin and a slight cyanosis of the lips. Eyes negative, nose negative, teeth in good condition. The throat was swollen and violently inflamed. Both tonsils and uvula covered with a dirty gray membrane. Breath foul. Cervical lymph nodes greatly enlarged. Lungs negative, heart negative, pulse rapid and thready. Blood pressure, 110-80. Abdomen negative, extremities negative, reflexes all active; 15,000 units of antitoxin were given and a culture taken from the throat. This was negative for Klebs-Loeffler bacilli, but contained staphlococcus aureus in pure culture.

Blood count showed: red, 3,800,000; hemoglobin, 70 per cent; white blood cells, 900; polymorphonuclear, 0; lymphocytes, 100 per cent. Urinalysis negative.

Aug. 3, 8 a. m.: Membrane has spread to the hard palate, over the gums and to the cheeks. Patient is breathing with considerable difficulty and pulse is very rapid and weak. There is a marked cyanosis. It is impossible to make a laryngoscopic examination.

Aug. 3, 2:30 p. m.: Patient is much worse, temperature 105°, pulse imperceptible at the wrist. Cyanosis is marked, breathing very labored and with marked stridor. Patient died while tracheotomy was being performed.

The literature is so replete with reports and discussions on the subject, as to whether the blood picture is an unusual result of absorption from the local ulceration, or the ulceration is a result of the blood condition, that no comment is made. Further study and early blood examinations in anginas may shed much light on this subject.

TUBERCULOSIS OF THE MAXILLARY SINUS; REPORT OF A CASE APPARENTLY CURED.

DR. EDLEY H. JONES, Vicksburg, Miss.

Tuberculosis of the maxillary sinus is an exceedingly rare disease. Gleitsmann, in 1907, reported that a search of the literature revealed only 20 such cases. The other sinuses are infected even less frequently.

The infection of the sinus is usually secondary to pulmonary tuberculosis, though it may be primary. Scheck reported such a case, the patient contracting the disease as a result of using her husband's handkerchief, he having pulmonary tuberculosis. Thompson stated that in general the tuberculosis is only an extension to the sinus from the bony wall or some neighboring focus.

Apparently very little has been worked out in regard to treatment. Kekwich, in 1895, reported using iodoform with good results. Schuster believes the treatment to be essentially surgical.

Aside from the rarity of the condition, the writer believes the following case report will be interesting because of an innovation in treatment—the use of chaulmugra oil, which apparently resulted in a cure.

Case Report: Mr. G. A. M., white, male, married, age 59 years, entomologist. *Chief Complaint:* Frequent small hemorrhages from right naris. *Family History:* Of no interest beyond noting that each member of the family enjoyed an unusually long life. No history of cancer, syphilis, tuberculosis, heart or kidney disease. *Past History:* Usual diseases of childhood. Has moved over the country a good deal, but has lived at present location for past six years.

Has suffered from kidney stones, passing first one from right kidney 26 years ago. Passed one from left kidney 11 years ago.

Had severe attack of Russian influenza 37 years ago. Dengue fever 12 years ago. Mild attacks of influenza eight and six years ago.

Two years ago daughter developed pulmonary tuberculosis, following influenza, and patient had, of course, been exposed. Daughter was sent to Texas, where she improved under treatment and was pronounced cured five months ago.

Four months ago patient was under a severe nervous strain, talking all day, every day for six weeks. "Hasn't been worth anything since". Patient has had no operations or accidents. Denies venereal history.

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Present Illness: Nov. 11, 1926, was awakened at 6 a. m. by blood trickling from right naris. Lost about ½-ounce of blood; controlled hemorrhage with cold packs. Worked all that day.

That night at 7 o'clock stooped over and blood began trickling again. Immediately he lay down and applied ice compresses to nose. Lost about 2 dr. of blood.

Same night, at midnight, awoke, bleeding again. Called family physician, who packed nose with adrenalin and prescribed Tinct. Ergot, teaspoonful each hour till bleeding stopped. The patient gave a history of bleeding freely, so the next day the doctor prescribed Elix. Chlorocalcium.

In spite of this, for past two weeks has had an average of one small hemorrhage per day, but never lost over an ounce of blood at any one time. Family physician referred him for examination and treatment.

Present Condition: Appetite fair; digestion good; has tendency towards constipation, but keeps bowels open with laxatives; sleeps fairly well, but coughs some through night. Uses tobacco moderately; no alcohol. Has been holding same weight for some time.

Physical Examination (by Dr. Otis Edgerton): Patient tall and slender; frame well developed but poorly nourished. Skin color good. Reflexes normal. Cervical glands slightly enlarged. Heart normal. Blood pressure, 130/85. Lungs show a well defined chronic bronchitis (patient has been a tubercular suspect for some time, but never able to make positive diagnosis). Abdomen negative. Extremities negative.

Oto-Laryngological Examination, Nov. 26, 1926: Ears negative. Nose: Septum normal; membranes slightly congested; ventilation good; no discharge; posterior rhinoscopy shows a small streak of old blood draining from the right side; transillumination negative; rhinopharyngoscope shows small streak of old blood draining posteriorly from under the right middle turbinate.

X-ray Report (Dr. Guy Sanderson): Nasal septum straight. Frontal sinuses fully developed, symmetrical and clear. Ethmoids and sphenoids clear. Right antrum shows a peculiar cloudiness at superior and inferior borders only. Left antrum clear.

Lungs show some scarring and some active tuberculosis.

Laboratory Reports: Blood hemoglobin, 70 per cent; red blood cells, 4,850,000; color index, .72; malaria not demonstrable; coagulation time, 4 minutes; leucocytes, 8,400; polymorphonuclears, 60 per cent; small lymphocytes, 28 per cent; large lymphocytes, 11 per cent; eosinophiles, 1 per cent; basophiles, 0.

Operation: Fearing malignancy, it was deemed best to open the right antrum for inspection. Accordingly, the antrum was opened Nov. 27, 1926, following Oertel's technique. Inspection revealed a tubercule, located on a transverse septum, extending downward from the roof about ¼-inch. The septum was removed and sent to the laboratory for examination. A large organized blood clot was removed from the floor of the cavity. The medial wall of the antrum was removed and the nasal moucous membrane flap from the inferior meatus was turned into the cavity and packed with iodoform gauze. The original incision was closed with sutures.

Progress Notes, Nov. 29: The packing was removed and a rubber tube inserted; 5 c.c. 10 per cent argyrol was injected through the tube daily.

Pathologist Report, Nov. 30: Tissue contained a small piece of bone, which appeared normal macroscopically, but was not examined microscopically. One or two areas of soft tissue gave some impression of tuberculosis, but there is not sufficient evidence microscopically to say definitely. The tissue shows definite chronic inflammatory changes.

Dec. 1. Examination of sputum; no acid-fast bacilli found.

Dec. 2. The patient was discharged from the Infirmary.

Dec. 6. Patient accidentally pulled tube out. Self-retaining tube inserted. Argyrol discontinued. Injected phenolated nasal (Sluder) oil daily.

Dec. 16. Removed rubber tube. Sent pus washed from antrum to laboratory, which reported "No T. B. (?), mixed infection". Sluder oil injected daily with a syringe.

Dec. 20. Washings from antrum again sent to laboratory, which reported "Positive T. B."

Using a silver cannula on a DH syringe, injected 5 c.c. chaulmugra oil in antrum. The patient was quite nauseated for the next two hours.

Dec. 22. Diluted chaulmugra oil to 50 per cent with liquid alboline. Injected 5 c.c. daily.

Jan. 18, 1927. No pus washed from antrum. Injections continued.

Jan. 25. No pus for entire week. Injections discontinued.

Feb. 24. Inserted rhinopharyngoscope in antrum; lesions appears healed.

Nov. 29. Patient reports that he has had no symptoms whatever that could be referred to right antrum. Rhinopharyngoscope inserted into antrum; no evidence of lesion other than healed scar. Patient reports that he had a general physical examination while in Texas a

few months ago and was told that his pulmonary condition had been arrested.

DISCUSSION.

Tuberculosis of the antrum is such a rare condition—much more rare than malignancy—that it was overlooked as a possible diagnosis even in the presence of pulmonary tuberculosis. On opening the antrum the clinical pathology was such a typical picture of a tubercule that the clinical diagnosis was easily made. It was a distinct surprise when the pathologist did not positively confirm this diagnosis.

When the drainage tube was removed, quite a bit of thick pus came out of it. This suggested another avenue of approach. The pathologist examined it carefully and found a few acid-fast bacilli. However, these were few in number and in the presence of a pulmonary infection he would not diagnose the antral discharge as tuberculous. Accordingly, he requested another specimen, which was collected three days later.

This second specimen was literally teeming with acid-fast bacilli. On the two slides, they were found in every oil-immersion field; sometimes they were found in clumps. There was no doubt that the exudate was tuberculous.

The question of proper treatment immediately presented itself. Recourse to the standard textbooks and treatises yielded nothing. Chaulmugra oil was experimentally used in treating tubercular laryngitis some years ago, but did not appear to be of any benefit. Knowing of no better treatment, the writer decided to try it. Chaulmugra oil "in vitro" effectively kills B. tuberculosis. Perhaps its use in an antrum more closely approximates laboratory conditions—a cavity, moist, nourishment and kept at a constant warm temperature—at any rate, it proved successful.

Use of the straight oil proved too severe, the patient becoming very nauseated and remaining so for two hours. Diluting it to 50 per cent proved satisfactory; he was sometimes slightly nauseated, but not unbearably so.

CONCLUSIONS.

1. Tuberculosis of the maxillary sinus is an exceedingly rare condition.
2. A radical antrum operation is indicated in such cases.
3. Fifty per cent chaulmugra oil proved of benefit in this case

312 First National Bank Building.

REPORT OF CASE, WITH PATHOLOGICAL SPECIMEN.*

DR. ISIDORE FRIESNER, New York City.

A boy, age 12½ years, entered the medical service at Mount Sinai Hospital with a history of an acute onset of fever nine days before admission and pain in the left ear two days later. The following day there was greenish-yellow discharge from the left ear. Temperature remained high, between 104° and 106°; no headache and no chills.

The boy presented the following clinical picture: Acutely ill, temperature 106.6°, pulse 144, respiration 34, flushed face, cyanosis of lips and dilating ala nasae. Heart sounds of good quality, regular. Systiloc murmur at apex transmitted to axilla. Liver and spleen felt two fingers below costal margin. Right ear normal. Left ear had slight purulent discharge, drum full posteriorly, canal wall normal, slight tenderness over the antrum. Hearing only moderately impaired. Blood count: W. B. C., 19,600; polys., 81 per cent; Hmgb., 75 per cent.

X-ray of mastoids showed cloudiness, with no evidence of breaking down of the septa. Fundi showed slight blurring of upper margins, otherwise negative. Blood cultures taken daily were negative.

During the following four or five days the temperature continued septic, the cyanosis became more marked, respirations were more labored. The evidence in favor of mastoiditis was meager, and the severity of the clinical picture could hardly be accounted for on the basis of an uncomplicated otitic infection. He became drowsy on the afternoon of the day of operation when his mastoid was opened and only a few discreet scattered droplets of pus were seen, without bony destruction. The sinus was uncovered but appeared normal. Pus from mastoid grew hemolyticus sterptococcus. Eight hours after operation the boy ceased. Blood culture taken day before operation, subsequently positive, hemolytic streptococcus.

The postmortem findings were: Brain edematous, dura normal. In the sigmoid sinus about 8 m.m. above the bulb there was a soft, friable, parietal thrombus firmly adherent to the inner wall, not completely occluding the lumen. There was also present an osteomyelitis of the petrous bone.

The heart showed a healed rheumatic endocarditis of the mitral valve and Aschoff bodies in the musculature.

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A boy, age 9 years, was admitted to Mount Sinai Hospital on March 16, 1927, with a history of discharging ear and pain in the left eye of two-and-a-half weeks' duration. Signs and symptoms of an acute mastoiditis, and a simple mastoidectomy was performed. In only small area of mastoid in the cells behind the sinus was pus present. Patient ran a very good postoperative course, the temperature ranging between 99° and 101° 17 days after operation. On the tenth day, P. O., however, patient complained of pain in left eye and in 24 hours developed an external rectus paresis on that side, which at the end of 48 hours became complete. The pain in the eye subsided for 48 hours, only to recur at intervals neuralgic in type. On the seventeenth day, P. O., patient suddenly had a rise of temperature up to 103½° and developed signs of meningitis, *i. e.*, stiff neck, bilateral Kernig. (Lumbar puncture showed streptococcis.)

A laminectomy was performed by Dr. Elsberg under general anesthesia and when the dura and arachnoid were incised there was a profuse escape of turbid cerebrospinal fluid under considerable pressure. At least 30 to 40 c.c. of fluid were evacuated. Streptococcus hemolyticus was recovered from the culture of the spinal fluid on repeated examinations. This was the same organism as that found on culture of the pus from the mastoid. Spinal fluid also showed 940 W. B. C. per c.c.m.

It is very interesting to note that on the following day after the laminectomy, the child did not complain of headache, rigidity of neck was distinctly less, and the paralysis of the external rectus was decidedly improved.

From that time onward the child became progressively worse, headaches returned, he vomited, became restless, irritable, had twitchings and convulsions until date of expiration, five weeks after original mastoidectomy, or about two-and-a-half weeks after laminectomy.

Postmortem Findings: Dura was found to bulge, due to a collection of fluid underneath it. The surface of the brain appeared to be injected. A small hemorrhage was present posteriorly along the falx. A thick, greenish, purulent exudate was seen over the basal surface, especially marked in the interpeduncular space and along the Sylvian fissures.

Pus present in cavum tympani, sigmoid sinus negative. *The apex of the left petrous pyramid was eroded and was covered by granulation tissue.*

Diagnosis: Osteomyelitis of apex of left petrous bone with destruction. Fibrinous purulent leptomeningitis and laminectomy. Purulent meningoencephalitis (streptococcus hemolyticus); purulent ependimitis.

36 East 73rd Street.

SUPPURATION OF THE PETROUS PYRAMID: RUPTURE OF EXTRADURAL ABSCESS OVER THE APEX: SUPPURATIVE CEREBRAL LEPTOMENINGITIS: DEATH. AUTOPSY.*

DR. RICHARD TRAVIS ATKINS, New York City.

Male, age 7 years. Admitted to the Harbor Hospital, April 5, 1927. Eleven days prior to admission the patient developed an acute otitis media on the left side, following an acute nasopharyngitis. The drum membrane was promptly incised. The ear drained profusely and there was a moderate elevation of temperature. Three days after the onset, a painful swelling developed in front of the ear.

On admission to the hospital, the temperature was 101.4° F., pulse 120, and respiration 28. There was a marked tender edematous swelling in front of the ear, which extended up into the scalp. Movements of the jaw were painful and markedly limited. There was mastoid tenderness; profuse purulent discharge; and narrowing of the canal at the fundus. The leucocyte count was 21,300, of which 76 per cent were polymorphonuclears.

A simple mastoidectomy was performed. The cells were of the large pneumatic type. They were generally filled with exudate, and there was free pus in the tip and suprasinus region. There was a large cellular zygomatic recess which was filled with pus; and at the junction of the middle and posterior roots of the zygoma a perforation was found, which lead into a localized abscess, from which an ounce of pus was evacuated. No dura or sinus was exposed. A counter opening was made in front of the ear and a rubber dam drain inserted. Culture of the pus from the mastoid showed a growth of hemolytic streptococci.

Following the operation the swelling completely subsided; the patient was apparently making a good recovery, until, on the tenth day following the operation, he began to complain of pain in the left eye. The pain continued for the next few days, and occasionally he also complained of pain in the teeth and the left side of the face.

The temperature oscillated between normal and 101° F. The leucoycte count was 26,500, of which 77 per cent were polymorphonuclears. The urine contained a faint trace of albumin; a few hyaline, granular and mixed casts, and at times there were a few pus casts.

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The eye grounds were negative.

Later, three weeks following the mastoid operation, the patient complained of double vision, in addition to the pain, and soon afterward developed a paralysis of the left sixth nerve. At this time there was also a sudden rise of temperature to 106° F., which was not preceded by a chill. The patient was nauseated but did not vomit.

The leucocyte count was 11,000, of which 62 per cent were polymorphonuclears. Culture of the blood was negative. The cochlea and semicircular canals were active.

The middle ear, which had previously been dry, now began to discharge again.

Because of the temperature reaction and the sixth nerve paralysis, an exploratory operation was performed on the lateral sinus. The internal jugular vein was ligated, and the lateral sinus opened and packed. There was no demonstrable clot found.

Following the operation, the temperature dropped to 101° F., and the patient seemed more comfortable. The pain was less severe and the paralysis less marked.

Later, however, the pain returned; and the patient also complained of some pain in the right side of the face and right eye. He had restless nights, due to the pain and headache. There was some pain and numbness of the legs.

The temperature ranged between 100° and 103° F. Culture of the blood still remained negative. The leucocyte count was 25,800, of which 70 per cent were polymorphonuclears.

As it was thought at this time that the patient had an epidural collection of pus in the middle fossa, another exploratory operation was performed. This consisted of an exposure of the dura of the middle and also the posterior fossae. There was no pathology found.

The patient was being transfused every other day, with no apparent beneficial effect.

Neurological examination by Dr. Foster Kennedy revealed the following: Pupils brisk, discs clear, no swelling. No V or V1 palsy. Kernig's sign present R>L. All reflexes normal. No sign of brain abscess. Meningitis is of dura and quite well localized.

Leucocyte count, 30,000, 82 per cent polymorphonuclears.

The temperature now remained fairly high, 102°-105° F.

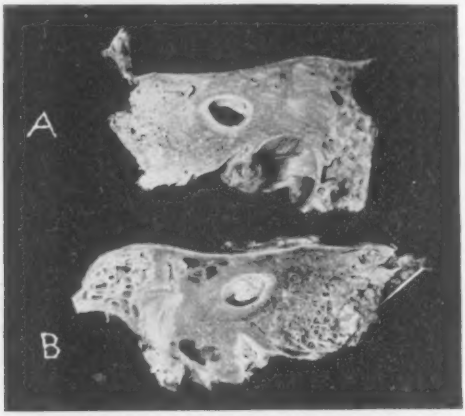
The patient complained of pain in the neck and back. He was irritable and very restless.

Later Dr. Kennedy reported as follows: Kernig more marked. No sign of brain abscess. There is threat against the meninges but all present symptoms are compatible with miningisinus and sinus thrombosis.

Another operation was performed by Dr. Alfred S. Taylor, one month after primary operation. The posterior and middle fossae were explored but no collection of pus was found.

The next morning the patient suddenly became cyanotic; lapsed into coma; and died.

An autopsy was performed by Dr. Wilder Penfield and his report is as follows: The body is that of a slender, well developed boy of about 10 years. The extremities and abdomen are normal in appearance. There is an incision in the left side of the neck which runs 12 c.m. upward and back of the left ear. There is another incision which passes posteriorly 5 c.m.



A. Left petrous pyramid, showing necrotic area at tip.
B. Normal right petrous pyramid.

The pupils are unequal, the right being about 2 m.m. in diameter, and the left, 4-5 m.m. They are even in outline.

Scalp: The hair is normal in distribution and appearance.

Skull: The skull is normal in shape, not unusual in size or thickness. There is a large operative defect in the skull at the left mastoid that extended back over the posterior fossa and forward into the middle fossa. The opening in the bone is about 7 c.m. anteroposteriorly and 5 c.m. from above down. There is a considerable amount of gauze packing between the bone posteriorly and the dura. There is no unusual evidence of bleeding in the operative wound. The left petrous pyramid shows no evidence of osteomyelitis, but at its tip beneath the dura, as will be described below, there is pus.

Dura Mater: Over the vault the dura is normal in thickness. No unusual herniations are present and there is no adhesion of the arachnoid to the dura, even in the left middle fossa. As the brain is removed, however, there is cloudy fluid evident in the left side of the middle fossa, although there is no unusual amount of fluid in the right. The horizontal sinus on the left contained some gauze packing and is thrombosed. There is a thrombus in the inferior petrosal sinus, but at the jugular bulb fluid blood is obtained. On dissecting off the dura from the summit of the petrous pyramid no superior petrosal sinus is seen on either side. As mentioned above, when an instrument was placed upward on the dura at the very tip of the petrous pyramid pus could be expressed from the dura here before any incision was made. The dura anteriorly over the tip of the pyramid is very much thickened and a small amount of pus is found here, forming an extradural abscess, which, however, seems quite small in amount. There is no evident neccrosis of the bone. Most of the horizontal sinus in the dura and of the posterior fossa is removed by the brain. The dura is dissected off of the petrous pyramid, except at its tip, where some of it is left. The whole petrous bone is then removed with the sella turcica. The opposite petrous bone is also removed.

On opening into the sphenoid sinuses no pathology is encountered. The nasopharynx is negative.

Leptomeninges: 1. Appearance: The meninges at the base are whitish, although in the midpoint over the interpeduncular cistern one can see through the arachnoid. There is obviously exudate in the fissure of Sylvius on the left side and a small amount on the right side.

2. The Cerebrospinal Spaces: The cisterna magna is full of pus. The subarchnoid space of the cord all the way down to the cauda equina contains purulent-looking fluid. In the basal cistern there is a white exudate and here in the interpeduncular cistern one gets the impression of organized exudate. Some of the intergyral fissures appear white, but for the most part they are clear. The left foramen of Luschka is wide open. The right could not be seen.

3. Pial Veins: There is not an unusual amount of congestion of the superficial vessels.

Brain: In color the brain does not appear unusual, excepting that the left temporal lobe is rather more yellow than the other. No softened areas could be seen. The convolutions are somewhat flattened, particularly over the left temporal lobe and the right hemisphere. The ventricles are not dilated and the ependyma and choroid are of

normal appearance. No exudate can be seen grossly on the walls. Sections through the brain show nothing abnormal.

Spinal Cord: It appears congested. The pia arachnoid seems a little opaque.

Gasserian Ganglion: Not removed.

Pituitary: Left in sella attached to the left pyramid of the petrous bone.

MICROSCOPIC EXAMINATION.

Tissue was taken for study from the left temporal lobe, cerebellum, pons and spinal cord.

Nerve Cells: The nerve cells throughout appear normal.

Oligodendroglia: These cells show swelling throughout, both in the satellites and those in the white matter. This was probably present for a short time before death, as it is rather too extreme to be purely postmortem.

Microglia: Not stained.

Astrocytes: In the cerebrum the astrocytes show rather marked clasmatodendrosis. The breaking up of these cells may occur postmortem also. Although it is rather extreme, it probably began before death.

Blood Vessels: The blood vessels appear normal. In general, there is no perivascular infiltration.

Pia-arachnoid: The pia-arachnoid over the pons and spinal cord is filled with leucocytes. Most of these are mononuclears.

There are a few polymorphonuclears. The same is true in the subarachnoid over the cerebral cortex.

In general, the brain shows only such changes as might have occurred shortly before death and as the result of an autolytic process. There is no gliosis or any other evidence of a chronic lesion of the brain. The meninges are filled with mononuclear leucocytes, which suggest that they came from a chronic abscess. Had the meningitis been of long duration one might expect more perivascular cuffing within the brain itself.

Note: An extradural abscess over the apex of the petrous pyramid has evidently ruptured and entered the basal cistern, finding its way from this point throughout the leptomeningeal spaces. The primary process seems, therefore, to have been the extradural abscess, associated evidently with an osteomyelitis of the petrous bone.

ANATOMICAL DIAGNOSIS.

Osteomyelitis of the petrous bone. Extradural abscess. Suppurative cerebral leptomeningitis. Thrombophlebitis of the inferior petrosal sinus.

4 West 53rd Street.

REPORT OF A CASE OF SUPPURATIVE OSTEITIS OF THE PETROUS PYRAMID.*

DR. EDWARD L. PRATT, New York City.

This paper is the report of a fatal case of meningitis caused by a suppurative osteitis of the petrous pyramid occurring nearly a month after an apparent resolution of a mild O. M. P. A.

I would *especially* direct attention to the "history" of this case and to the absence of any definite otological symptoms or physical signs prior to the time when the physical signs suggested a diagnosis of meningitis.

History: The patient was a robust boy, age 5 years, who had always enjoyed excellent health. On Aug. 5, 1927, he went in bathing. That evening he complained of slight discomfort in his right ear. He apparently had no *severe* pain in the ear, as he slept all night. The next morning his mother noticed that his pillow was stained and that the boy had a profuse serosanguinous discharge coming from his right ear. The family physician was summoned and boric acid irrigations were ordered. The discharge was rather profuse for the next two days, but rapidly diminished in amount until, on the *fifth* day after the onset, the ear was dry.

During this period the boy showed no signs of being acutely ill. He was kept indoors the first three or four days, but not in bed. He had no complaints; no elevation of temperature and was as alert and active as in normal health.

On Aug. 13, *eight days* after the onset of the O. M. P. A., he motored to Massachusetts with his family and remained there for *two weeks*. During these two weeks there was no discharge from the ear but, on several occasions, he complained of pain, which he described as "in the ear". These attacks, in the beginning, occurred only once or twice a day, lasted only a few moments, and were considered by his parents as of no significance.

On Aug. 28, the family returned to Rye after a three-day motor trip from Massachusetts. The boy then appeared acutely ill and complained of *continuous* pain in his right ear and tenderness on pressure behind the ear. Two days later the family physician was consulted. Physical examination was negative and the patient was

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referred to me for an otological report. This was the first time I had seen the case—27 days after the onset of the O. M. P. A.

The child looked acutely ill and appeared to be rather somnolent and somewhat stuporous. He responded to questions intelligently but was slow in answering and appeared quite indifferent. His temperature was 102°. Examination of the nose and throat was negative. There was no discharge in either auditory canal and *both* membrana tympani were normal in color and contour. There was no evidence of diminished acuity of hearing in either ear. Schwabach test was normal in both ears; Rinné positive on both the right and left side; no lateralization in the Weber test; no spontaneous nystagmus; no vertigo. He was moderately tender all over the right side of the head, but *without any localized tenderness.*

I reported these negative findings to his physician and recommended a blood count, a Widal and X-rays of both mastoids. The blood count showed 12,700 white cells with 74 per cent polys. The Widal was negative. The mastoids were *not* X-rayed.

He was kept in bed and was seen daily by the family physician. His temperature ranged between 101° and 102° and he continued to complain of pain "in the ear". His physician recorded on the chart that "the boy does not appear to be acutely ill. He is somewhat stuporous but otherwise his mental state appears normal. He talks, plays, and is interested in stories and picture books. His appetite is excellent."

There were no positive physical signs until three days later, when a slight rigidity of the neck was noted. A lumbar puncture disclosed turbid fluid with slightly increased pressure. Cell count, 9,600; polys., 92 per cent; lymphocytes, 6 per cent; mononuclears, 2 per cent. Sugar reduction absent; globulin, $\pm$, culture; hemolytic streptococci. Examination of the fundi showed blurring of the disc margins and enlargement of the veins; no elevation was made out. Ocular movements and vision normal.

I was again asked to see the case, and again my otological findings were negative. Both membrana tympani appeared normal; there was no discharge from either ear, and his hearing tests were normal. X-rays were then made of the mastoids but they showed no *definite* evidence of any inflammatory involvement. The right drum was incised but no fluid was found in the middle ear.

The acute otitis media of Aug. 5 appeared to be the only portal of entry which might account for his meningitis, so it was decided that an exploration of the right mastoid was warranted.

On Sept. 4, 1927, 30 days after the onset of his O. M. P. A., I explored the right mastoid at the United Hospital in Port Chester.

All the mastoid cells were exenterated, but no pus was found, nor was there any evidence of suppurative involvement of the mucosa of the cells *except* in the region of the tegmen antri. Here the membrane lining the cells was thickened and hemorrhagic and the bone appeared to be somewhat softened, but not definitely necrotic. A portion of the plate over the middle fossa in this region was removed but the dura appeared to be normal.

Subsequent Course: For four days following the operation there appeared to be a decided improvement in the *mental* condition of the patient. He was less stuporous and seemed to be brighter, more alert, and more interested in his surroundings. There was no improvement in his *general physical condition,* however. His temperature ranged between 102° in the morning to 104° in the afternoon. His spinal fluid was more turbid, the cell count increased, and the rigidity of his neck was more marked. He then became more and more stuporous and on Sept. 13 was in a state of coma. Dr. H. A. Riley was called in consultation. He found no evidence of any focal lesion; no change in the optic discs since the previous examination; reflexes equal. He advised further exploration of the middle fossa and a blood transfusion.

A few hours later I uncovered all of the mastoid dura of the middle fossa, incised it, and explored the brain, with negative results aside from the escape of cerebrospinal fluid. The sigmoid sinus and contiguous dura was also exposed, but no pathological lesion was found. A transfusion was given.

Following this secondary operation there was again a decided change in the *mental* condition of the patient. Before operation he was in a state of coma; for the next 48 hours he was conscious and rational, but there was no change, however, in his *physical condition,* and he again became stuporous, lapsed into coma, and died four days later. The middle ear was dry throughout his illness and there were no focal signs until a few hours before he died, when a left external strabismus and spontaneous nystagmus of both eyes was noted.

Autopsy Report: The autopsy, which was limited to the head, was performed by Dr. Morton Ryder, of Rye, N. Y., to whom I am indebted for the following report:

"Anatomical diagnosis: *1.* Acute suppurative generalized cerebrospinal meningitis. *2.* Necrosis of the petrous portion of the right temporal bone on its anteriosuperior aspect. *3.* Operative removal of right mastoid cells.

"Calvarium removed in usual manner. Dura was quite adherent over vault and base. Dura normal except for granulations over areas

exposed at operation. When dura is opened there is a gush of thin, turbid fluid. Convolutions of both hemispheres are slightly flattened; vessels are moderately engorged and there are collections of pus along the larger vessels, especially in the Sylvian fissures. Base of brain is covered with a fibrinopurulent, yellowish mass of exudate which is *very extensive,* is several millimeters in thickness and almost completely hides the brain stem. All the ventricles are filled with purulent material, which escaped when the brain was removed (not measured, but certainly several ounces). Choroid plexus and lining of ventricles congested and adjacent brain tissue softened. Section of entire brain reveals no focal lesion. Nasal accessory sinuses normal.

"On the anterosuperior aspect of the petrous portion of the right temporal bone there is a shallow area of necrosis about 1 c.m. in diameter and 1 or 2 m.m. in depth; outlines irregular. Beneath the base the bone is dark and soft. The center of this area lies at about the junction of the lateral third and medial two-thirds of the petrous portion of the temporal bone. There is a narrow tract leading from this area to the antrum."

29 East 64th Street.

TWO CASES OF PETROUS BONE ABSCESS DRAINAGE; RECOVERY.*

DR. WESLEY C. BOWERS, New York City.

Reviewing the cases of petrous bone abscess, if we count only those which have been verified by autopsy or operation, the number will be few. In actual fact, however, they are far more common than we, in the past, have suspected. The majority of the cases with Gradenigo's syndrome, most of those showing fifth nerve pain without involvement of the abducens, many of the cases which suddenly develop meningitis, possibly weeks after a mastoidectomy, and some of the cavernous sinus involvements are undoubtedly due to pathology in the petrous bone. I have seen a considerable number of cases which were undoubtedly due to involvement of the petrous bone (most of which died with a fulminant meningitis). These, however, were not so proven by autopsy or operation and are therefore valueless from the standpoint of case reports.

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The report of the following operated case is given in some detail as illustrating some typical symptoms, as well as a mode of attack and a method of drainage which I have employed successfully in two instances.

J. M., age 6 years, suddenly developed pain in both ears. Bilateral myringotomy was promptly performed. Aural culture taken at the time of myringotomy showed pneumococcus Group 3. The ears stopped discharging in about 10 days; in about two weeks the hearing and drum membranes had returned to normal. Nineteen days later, a facial paralysis developed on the right side. Examination at this time showed both drums normal; hearing normal, Weber not lateralized; rotation gave a reaction of 20″ in both sides; the eyes were negative; all reflexes normal; taste sensation doubtful because of poor co-operation. X-ray showed the right mastoid cells sharply outlined but slightly cloudier than the left. Because the infecting organism was pneumococcus Group 3 and because of the right facial paralysis, the right mastoid was operated upon.

All cells were filled with granulations; the bone was soft in places, including a small area over the dura in the middle fossa; there was no free pus. The antrum was filled with granulations which seemed to be unusually dark and tough. Culture from the wound was sterile.

The postoperative history was as follows: First day, with normal temperature and good hearing, there was a very slight nystagmus to the right. Second day, normal temperature, good hearing. The patient complained of slight dizziness at times and vomited food almost immediately after taking it. There was no nystagmus. Third day, temperature normal and hearing good. A spontaneous nystagmus to the left developed, with marked vertigo—much increased on turning to or lying on left side. The facial paralysis was considerably improved. The blood count was practically normal. Fourth day, no temperature, hearing good, Weber unsatisfactory because of poor co-operation. The drum membrane, which *up to this time had been normal*, showed slight bulging, with some discharge coming from it. The spells of vertigo were very marked, terrorizing the patient; these were accompanied by very severe nystagmus to the left, lasting half-a-minute; there was emesis on taking food, but not with the vertiginous attacks. There was no rigidity, no headache, normal reflexes. Fifth day, no temperature. Whisper heard with noise apparatus. The drum membrane remained the same, the vertigo was much improved, the nystagmus unchanged. Sixth day, the temperature was 100°. The hearing was good, there was slight aural

discharge, and some pain in the mastoid region. Nystagmus was the same; hot caloric gave no reaction. The blood count show: W. B. C., 11,900; polys., 68 per cent. Seventh day, temperature 99°. Hearing gone. The blood count showed: W. B. C., 13,000; polys., 66 per cent. There was some headache, no rigidity; the facial paralysis had entirely cleared up. There was some pain in the wound. Eighth day, the temperature was 101.2° following a restless night, the nystagmus was unchanged, there was slight Kernig and stiffness of neck, eyes negative, blood count about the same. Spinal fluid showed 360 cells, protein 4 plus, culture negative.

What we were apparently facing at this point was an acute suppurative labyrinthitis with beginning meningitis; the facial paralysis had cleared up, the middle ear had begun to discharge and the vertigo was not of the labyrinthian type.

A radical mastoid operation was performed. Unhealthy granulations were present in the mastoid cavity; the middle ear was filled with granulations. While following some cells in front of the superior semicircular canal, above the facial, a cavity was entered from which came considerable pus. This cavity was about ½-inch in diameter, extending downward and inward in the petrous bone, above, behind and below the facial nerve, and internal to the labyrinth. The dura was exposed by the abscess for an area of ½-inch on the anterior surface of the petrous bone. While cleaning out some cells below the posterior semicircular canal, the abscess cavity was again entered. Both openings were enlarged as far as possible and rolled rubber dam drains inserted. The labyrinth was not opened externally as, in my opinion, the abscess in itself was sufficient explanation of all symptoms. Undoubtedly, both the auditory and facial nerves were exposed in this cavity and very probably the labyrinth connected directly with it. Culture from the abscess cavity showed pneumococcus Group 3. It is especially noteworthy that culture from the original myringotomy showed pneumococcus Group 3, culture from the mastoid wound was sterile, and culture from the petrous bone abscess showed pneumococcus Group 3.)

The child made an uneventful recovery. The rubber dam drains were undisturbed until they came out of themselves, after which no attempt was made to introduce anything into the cavity. Eighteen days later, the nystagmus had disappeared; the hearing was, of course, still gone. One month after the radical operation, rotation to the right gave a reaction of 10 seconds, to the left, 6 seconds. All other tests were normal. The patient was last seen 12 months after the operation, at which time the radical cavity was dry and the patient normal in every way.

I have had one other case, also with a dead labyrinth and beginning meningitis, in which drainage of a petrous bone abscess, by the identical method and in the same place and way, was followed by recovery.

Points to Be Considered: *1.* Pneumococcus Group 3 in aural discharge; function and appearance of ear returning to normal; sterile culture from mastoid wound 21 days late; pneumococcus Group 3, subsequently, in petrous bone abscess. These findings would indicate that nature had succeeded in overcoming the infection in the mastoid, but had failed in some cells deep in the petrous bone, the result being the formation of an abscess beyond the labyrinth, which involved the facial and auditory nerves.

2. The child had no temperature until meningeal symptoms developed, despite all the symptoms of a labyrinthitis and the presence of an abscess ½-inch in diameter, in the petrous bone. The highest blood count was 13,000 white cells; polys., 66 per cent. The drum membrane was normal, the hearing normal; yet there were curiously dark, tough granulations in the antrum.

3. These cases illustrate very plainly that it is absolutely necessary to do a radical mastoid operation in cases of beginning meningitis where nothing to account for the symptoms has been found in the mastoid region, with the hope of being able to locate an abscess in the petrous bone, by following the cells in front of the superior semicircular cana, above the horizontal portion of the facial nerve; also, below the posterior semicircular, behind the vertical portion of the facial. The portion above the horizontal portion of the facial is the most likely point of encounter of such an abscess (perhaps epidural), in those cases which complain of pain in the temple or the eye. The portion below the labyrinth, in the region of the ductus endolymphaticus, is the most probable location of an abscess in cases with labyrinthian symptoms. In the given case, drainage was re-established in both the above locations. The rubber dam drains were left in place until they came out of themselves, and nothing reinserted. The ensuing, uneventful recovery demonstrates that an abscess in the petrous bone may be drained successfully in this manner.

17 East 61st Street.

LABORATORY AIDS IN OTOLOGY.*‡

DR. SAMUEL J. KOPETZKY, New York City.

Otology, during the past twenty years, has been greatly enriched by the products of research. From a narrowly restricted field it has so broadened that it now encroaches upon several of the specialties to which it formerly bore only a remote relationship. In its growth, it was only natural that the confusion and awkwardness of adolescence should accompany the new lore. Now that it has passed that stage, it is imperative that it classify its knowledge, cross-index the new with the old and discard the worthless. We will then be ready for further additions, which, like the ordered parts of a mosaic, can be fitted into their proper places.

While otological surgery has progressed remarkably during these years, it is in the utilization of the numerous advances in laboratory medicine that an even more remarkable progress has been made, particularly in the field of diagnosis. But here the cross-indexing is not yet complete; and I propose, by correlating the laboratory aids at our disposal with our already well indexed clinical data, to clarify some of the confusion which is manifest.

The diagnosis of the surgical diseases in otology has far surpassed our ability to recognize the non-surgical conditions. It is therefore evident that the laboratory has aided us more accurately in the recognition of the former types of lesions. By study of the blood, the cerebrospinal fluid, the bacteriological flora and the Roentgenographic pictures of the mastoid process, clinical diagnosis has been rendered a great deal more precise in surgical otology. In the nonsurgical diseases of the ear, while laboratory studies have as yet yielded scant information, here too great promise is held forth. I shall speak first of the laboratory aids in the diagnosis of the surgical diseases of otology.

Blood Studies: The routine estimation of the *red cell count* has not been accorded the position its importance warrants. I have found it of great diagnostic value. Examination of the red cells, however, is never complete without an estimation of the hemoglobin percentage. The two go hand in hand as a diagnostic aid, both

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before and after an operative procedure. I have repeatedly emphasized the distinction between the two types of acute mastoiditis, the acute coalescent mastoiditis and the acute hemorrhagic mastoiditis. The former is the one most generally encountered. There are a breaking down of the intercellular walls, a coalescence of the cells and the formation of pus and granulations. The hemorrhagic type is more rare. It is characterized by a multiplicity of thrombi in the minute venules of the lining of the mastoid cells. In this type we never see a coalescence of the cell walls with a consequent formation of pus. The lesion, no matter what its stage at operation, always exhibits intact cell walls. Clinically, the marked tenderness over the mastoid process, the severe prostration and the septic temperature presented at the onset of the ear lesion are never in accord with the otoscopic picture, where no sagging of the canal wall and no profuse discharge are evident. Those of you who have seen such cases during the late influenza epidemic will recall what were considered to be unusual findings at operation. But the sharp recovery after operation on the mastoid process warranted the continuance of surgical intervention in these cases, although they presented none of the classical signs of acute mastoiditis.

Bacteriological investigations showed this type of mastoiditis to be due to the streptococcus hemolyticus. It is in the hemorrhagic mastoiditis, particularly, that repeated examinations of the red cells and the hemoglobin percentage will result in early recognition of the lesion. In a patient who presents an acute purulent otitis media, accompanied by septic temperature and severe prostration from the outset of the disease, a daily decrease in the number of red cells and in the percentage of hemoglobin speaks for a diagnosis of acute hemorrhagic mastoiditis, and indicates an immediate simple mastoidectomy where all other conditions have been eliminated. Since the lesion in this type of mastoiditis is present in the small vessels within the mastoid process, the hemolytic streptococci present within the infected thrombi destroy the hemoglobin and the red cells of the blood stream. This is evidenced by the gradual fall in the number of red cells and in the percentage of hemoglobin.

After a simple mastoidectomy in this type of mastoiditis, thorough exenteration of the mass of infected thrombi is followed by a subsidence of the sepsis and a gradual return of the red cells and hemoglobin to normal. Should the reduction in the red cells and hemoglobin continue after the simple mastoid operation, a further extension of the disease into the lateral sinus should be suspected, and a transfusion of the whole blood is in order. If the reduction still

continues, the lateral sinus should be obliterated and the internal jugular vein ligated regardless of whether or not the outer wall of the sinus is diseased. Since the sinus thrombosis in this type of mastoiditis is produced by the intravenous extension of the minute thrombi directly into the lateral sinus, there will rarely be found any erosion of the sinus plate or any evidence of a periphlebitis. In this type of mastoiditis, continued reduction of the red cells and the hemoglobin after operation furnishes a better diagnostic aid than the appearance of the sinus wall.

In the acute coalescent mastoiditis, a daily study of the red cells and hemoglobin throughout the period of hospitalization subsequent to operation will always lead to an earlier detection of a sinus thrombosis where a hemolytic organism is the offending factor. In these cases, the earliest appearance of a thrombosis will be manifest in a gradual daily decline in the number of red cells and the percentage of hemoglobin. Where the hemoglobin falls steadily, and where the clinical signs of sepsis are absent, a transfusion will replace the lost hemoglobin and so add to the patient's resistance. On the other hand, a persistent unilateral headache after operation, accompanied by an increased leucocytosis and a gradual fall in the number of red cells and the percentage of hemoglobin, should lead one to inspect the interior of the sinus for the presence of a thrombus. The utilization of the red cell and hemoglobin estimation as a routine in every case of mastoiditis will afford an early guide in the detection of otitic sepsis, whether it be due to an acute hemorrhagic mastoiditis or to a complication of an acute coalescent mastoiditis or to a chronic mastoiditis.

The White Cell Count: The study of the *white cells* furnishes us with a diagnostic as well as a prognostic aid. In the ordinary white cell count, the otologist is interested in the total number of white cells and the percentage of polynuclear cells. The former is the index of the resistance of the individual while the latter indicates the severity of the infection. Utilizing this as a foundation, McKernon drew up a chart whereon the total number of leucocytes could be compared with the percentage of polynuclear cells. He determined that with a given number of leucocytes a patient could present a certain maximum percentage of polynuclear cells before his resistance was overcome by the virulence of the infection. Once this was determined, we had at our disposal an excellent prognostic guide. Knowing the maximum number of polynuclears that is compatible with any given leucocytosis, we can determine at a glance whether or not the patient has sufficient resistance to combat the infection.

Where the percentage of polynuclears exceeds the maximum allowed for any given number of leucocytes, the patient's reistance is succumbing to the infection. On the other hand, where the polynuclear percentage is less than, or equal to, the maximum allowed for any given total leucocytosis, the resistance of the patient is good. The use of these resistance charts will be found of inestimable value in prognosticating the outcome of any otitic infection.

The white cell count is also of value as an indication of an impending complication. After a simple mastoidectomy, the white cell count should show a rapid decline toward normal. A persistent leucocytosis with an increase in the percentage of polynuclear cells is indicative of a continued infection. A careful observation of the patient is necessary in such cases to determine the source of the infection that is causing the leucocytosis to persist. Most frequently it is due to the continuation of purulency in the middle ear; and an increased white cell count may be noted until the middle ear infection has subsided. If, however, the increased leucocytosis is accompanied by a unilateral headache, an extension of the disease beyond the mastoid process should be suspected. Where, following operation, the leucocytosis subsides, only to recur after an interval of time, accompanied by unilateral headache, a sinus thrombosis should be strongly suspected. I have had several cases of sinus thrombosis subsequent to the acute coalescent mastoiditis wherein the only signs that led to a diagnosis of sinus thrombosis were the persistent unilateral headache and the increased leucocytosis. In these cases I had no other signs or symptoms of sepsis on which to base a diagnosis.

Recently I have had experience with the method of counting the white blood cells devised by von Schilling and introduced at Beth Israel Hospital by Dr. Arthur Weiss. In brief, the count is made as follows: The total number of cells is tabulated as heretofore. In the differential count, however, particular attention is paid to the various morphological forms of the polynuclear cells. One notes the percentage of mature polynuclears; that is, those in which the nucleus has undergone complete segmentation, and the percentage of young polynuclears, wherein the nucleus is still unsegmented and appears in the form of a rod. The latter type are called the "staff cells." In a normal count, the young forms, or staff cells, number less than 5 per cent. At the onset of an acute infection which produces a polymorphonuclear leucocytosis, there occurs a marked increase in the number of these immature staff cells. This is due to the fact that the bone marrow is stimulated to produce an excess amount of polynuclears to combat the infection and these cells are thrown into the

general circulation before their nuclei have had time to undergo complete segmentation. In addition to this, the percentage of lymphocytes diminishes. This picture has been termed a "shifting to the left." As the infection subsides, the number of staff cells also becomes less until "normal" is finally reached. The blood picture does not have any relationship to the temperature curve but is in direct relationship to the virulence of the infection and the resistance of the patient. Where, following a simple mastoidectomy, the number of staff cells continues high, that is, above 15 per cent, it is indicative of a continuation of the infection. It is not diagnostic of any complication but it should put the surgeon on guard to watch for the clinical signs of a complication. The continuance of a high staff count, or the reappearance of one after it has returned to normal subsequent to aural surgrey, should be viewed with the utmost suspicion.

I have found this method of counting the white cells of inestimable value in determining the procedure to be followed in the so-called borderline cases. Where a mastoiditis is presented and one is in doubt as to whether or not to operate, the staff cells will furnish an excellent guide in helping one to reach a decision. Where the staff cells increase in number on several daily examinations and reach 12 per cent or more, operation is indicated. On the other hand, where the percentage of staff cells remains stationary below 12 per cent, or shows a gradual reduction, operation can be postponed with safety and the patient kept under further palliative treatment.

Blood Culture: Before discussing the results of blood culture and the importance to be attached thereto, I should like to discuss as briefly as possible the pathogenesis of a sinus thrombosis. In the acute coalescent and the chronic mastoiditis, there occurs a gradual destruction of the mastoidal structure until the inner table is reached. The inner table overlying the sinus is then subjected to pressure from the pent up pus and undergoes necrosis. The outer wall of the sinus is thus brought into direct contact with the purulency within the mastoid process. The pressure against the wall of the sinus causes a narrowing of the blood channel with a consequent slowing of the blood stream. In addition, the outer wall of the sinus becomes infected by the pus in the mastoid process and we then have a sinus periphlebitis. If the mastoid is operated upon at this point, there is found a protective layer of granulations over the sinus wall at the exposed area. On the other hand, should this periphlebitis be permitted to progress so as to involve the endothelial lining of the sinus, the injury presented to this lining membrane, plus the narrowing of

the channel and the slowing of the flow of blood, offer all the elements necessary for the production of a thrombus formation. This thrombus is protective in nature and is at first sterile. If the round cell infiltration and granulations on the outer coat of the sinus are not sufficient to keep out the organisms from within the sinus, the thrombus will become secondarily infected and we then have an infected thrombus within the sinus. This thrombus grows with and against the blood stream and only its central portion is infected: its extremities are sterile. Therefore, if a blood culture is taken at this stage of the thrombosis, no growth of organisms will be noted. If nature is successful in localizing the infection by causing a fibrosis of the thrombus, a positive culture will never be obtained. It is only when the thrombus has undergone disintegration that infected particles will gain entrance to the general circulation and on culture of the blood a positive growth of bacteria will be obtained. To permit every sinus thrombosis to go on to this stage before operative intervention is undertaken is not in accord with the best principles of surgery. One does not wait for an acute suppurative appendicitis to rupture before removing it surgically. So, in otology, one must not view a negative blood culture as ruling out the presence of an infected thrombus within the sinus where the clinical symptoms point to its presence. Too much reliance has been placed upon the blood culture as a means of diagnosing sinus thrombosis. It has its value as a diagnostic measure but is not nearly as reliable in the greatest proportion of cases as the red cell and hemoglobin estimation and the white cell count. The findings at the primary operation on the mastoid process, coupled with the appearance of temperature, unilateral headache, etc., are also of far greater diagnostic value than the results of blood culture. In the last 18 cases of sinus thrombosis upon which I have operated, the blood culture was positive only in four of them. In two instances wherein I had obtained a positive blood culture of streptococcus hemolyticus in cases of sepsic subsequent to mastoid surgery, I excluded a sinus thrombosis from the clinical picture presented by the patients because of other factors. In both instances the patients recovered without resort to surgery on the blood vessel.

A positive blood culture is more apt to be found in cases of sinus thrombosis subsequent to the hemorrhagic mastoiditis. Here the thrombosis in the lateral sinus is the result of the direct growth of a smaller thrombus from one of the venules in the cellular walls into the larger vessel. The sinus plate is not eroded, no perisinal abscess is found, and there is no involvement of the outer coat. The throm-

bus in this instance is infected from the outset and is more often of the mural than the occluding type. The blood stream, in passing, washes some of the organisms into the general system, and a positive blood culture is obtained much earlier than in the thrombosis following the acute coalescent mastoiditis.

In addition, a negative blood culture is to be expected where metastatic lesions are present. These lesions, particularly if they are located in the soft parts, act as fixation abscesses and clear the blood stream of infection. If the bacteria gain entrance to the pulmonary circulation, they are apt to lodge there and produce their characteristic lesion without ever reaching the general circulation.

Blood Sugar: A blood sugar should always be part of the routine examination of any adult suffering from a mastoiditis. A diabetic who is under treatment will often show no sugar in the urine while estimation of the blood sugar will reveal a hyperglycemia. Diabetes produces that type of ear lesion which has been termed the painless type of acute coalescent mastoiditis. An extensive necrosis of the mastoid process may exist and yet the patient have no pain nor tenderness. Where the otoscopic picture and the duration of the disease point to a definite acute surgical mastoiditis but wherein, because of the absence of pain and temperature, one is inclined to temporize, the finding of an increased blood sugar is a warning of the danger that the diabetes presents. If such a case is permitted to proceed without surgical intervention, the pain and temperature will eventually appear concomitant with an intracranial lesion.

The Cerebrospinal Fluid: Lumbar Tap: A lumbar puncture is indicated in every case of chronic suppurative otitis media before operation is undertaken on the mastoid process. It is indicated in every case of postoperative simple mastoidectomy wherein an intracranial lesion is suspected. The information to be gained at the time of the tap is the amount of intraspinal pressure and the presence of a block between the intracerebral and the intraspinal subarachnoid spaces. The latter can be determined by noting the rise in manometric pressure following jugular compression. If the spinal fluid is permitted to run into a mercury manometer, the pressure of the spinal fluid can be read. If pressure is then brought to bear over the internal jugular vein, first on one side and then on the other, a rise is noted in the intraspinal pressure where no obstruction exists between the intracranial space and the spinal column. Incidentally, Tobey and Ayres have utilized this procedure in the differential diagnosis of sinus thrombosis in cases of bilateral mastoiditis. The side whereon the thrombus is present is obstructed and consequently com-

pression of the internal jugular on that side will cause no rise in the intraspinal pressure. I have utilized this procedure to great advantage and found it to be an excellent diagnostic measure. It is not infallible, however. Where a thrombus is not of the obliterating type, compression of the jugular will permit a rise in intraspinal pressure. Then again, in one of my cases wherein I had completely obliterated the lateral sinus after incision and resection of a portion of the outer wall, but wherein the internal jugular vein had not been operated upon, compression of the internal jugular vein on the side of operation caused a rise in intracranial pressure equal to the rise obtained from compression of the opposite internal jugular vein. This case has been reported by me elsewhere.

In cases of acute labyrinthitis following suppurative otitic disease, the spinal fluid will serve as a diagnostic measure in determining whether or not the labyrinthitis is of the suppurative type. Where the spinal fluid shows no increase in the number of cells, has a normal chemistry and shows but a slight increase in pressure, the labyrinthitis is of the serous variety. On the other hand, where a large increase in cells is noted and the albumin is increased, a suppurative labyrinthitis is to be suspected. If the increase in cells is predominantly lymphocytic, one can continue to employ palliative measures. Where the increase is largely polynuclear, a threatened invasion of the subarachnoid space is indicated and operation is advisable. This is especially true if, on successive taps, a high lymphocyte count is found to be replaced by a high polynuclear count.

If examination of the spinal fluid shows a high cell count, lymphocytic or polymorphonuclear, increased albumin and globulin and increased pressure, but no bacterial growth, one is dealing with a sympathetic meningitis. This is always indicative of a threatened invasion of the subarachnoid space. It can be produced by a sinus thrombosis, brain abscess or suppurative labyrinthitis. Where such a fluid is obtained, every measure must be employed to immediately eradicate the focus of infection, if a suppurative meningitis is to be averted.

One of the earliest findings in the spinal fluid in cases of suppurative meningitis due to a fermentative type of bacteria is a reduction in the percentage of glucose normally found in the spinal fluid. This is often demonstrable even before any organisms can be isolated. Where the copper reduction is absent or diminished, a suppurative meningitis can be diagnosed even though no bacteria can be isolated on smear or culture.

Bacteriology: A culture of the pus from the middle ear is of no value unless taken very soon after the paracentesis or spontaneous rupture of the drum. Cultures taken after twelve hours or so will almost always show a mixed infection due to contamination. A culture of a chronic aural discharge is of no value at any time because the organism isolated is usually different from that which produced the lesion originally.

There are only four organisms which when isolated from an aural discharge influence the management of a case: the Klebs-Loeffler bacillus, the Vincent's bacillus and spirillum, the streptococcus mucosus capsulatus and the Friedlander's bacillus. The bacillus of diphtheria naturally indicates specific treatment with antitoxin. The Vincent's bacillus and spirillum necessitate salvarsan. The other two organisms depict an extremely dangerous type of lesion, insidious in character and asymptomatic in its course. Because of the very nature of the disease, it often goes on to intracranial invasion with no clinical evidence of its progress. Hence the finding of the streptococcus or the bacillus mucosus capsulatus indicates early complete surgery on the mastoid process in order to forestall intracranial invasion. It is true that a number of cases wherein these organisms have been found recover without any operative measures, but the seriousness of the mastoiditis due to these bacteria must be considered from the standpoint of the high mortality presented.

Roentgenographic Examination: Roentgenographic examination of the mastoid process is to be considered an accessory laboratory aid in otological diagnosis. The otologist should be able to interpret the findings on radiographic examination of the mastoid himself. One cannot expect the Roentgenologist to make a clinical diagnosis from the plate; and the Roentgenologist who does so is presumptuous unless he is specially trained in otologic pathology. An X-ray examination will yield anatomical data and will show whether or not cellular destruction is present. It will not label the case as surgical or nonsurgical, acute or chronic, emergent or nonemergent. This the otologist alone can determine by a correlation of the clinical, laboratory and X-ray findings.

In every acute middle ear infection, a clouding of the mastoid process will be noted. This is due to the edema of the cellular lining which accompanies every acute middle ear suppuration. The same cloudiness will also be found in cases of external otitis accompanied by inflammation of the periosteum over the mastoid process. In the absence of any evidence of cellular destruction, the finding of a cloud-

iness of the mastoid process has no significance from a clinical standpoint.

Where the diminution in illumination is accompanied by evidences of cellular destruction in an acute middle ear infection, an acute coalescence of the mastoid cells is indicated. Here, however, the clinical appearance of the ear and the general systemic reaction will guide the surgeon in his determination as to whether or not the case is a surgical one. A certain amount of mastoidal destruction may exist and yet the case may recover without surgical intervention on the mastoid process. In such instances successive mastoid plates are valuable to gauge the progress of the destruction. In a patient who has had one or more attacks of mastoiditis prior to the current acute attack, the finding of cellular destruction on the mastoid plate may be the result of the previous mastoid inflammations. Here, also, repeated X-ray examinations are valuable in determining whether there is an extension of the destruction.

In a case which has been under observation for a greater length of time than that usually required for the cessation of an acute aural suppuration, the finding of an extensive cellular development of the mastoid process with a small amount of destruction would tend to indicate a beginning chronicity and operative interference is usually advisable before complete destruction occurs. On the other hand, the same amount of destruction in a small mastoid process would indicate that the infection was being walled off and would recede of its own accord.

Very frequently, on a well taken plate, an erosion of the sinus plate or tegmen can be noted by a break in the continuity of the shadow cast by these structures. In this way an epidural or perisinal abscess can be detected.

In the chronic purulent otitis media, a sclerosis of the mastoid process will be noted in every case. This is not to be interpreted as indicating a radical mastoid operation for it will be found in all cases —dangerous and nondangerous—of chronic purulent otitis media.

In taking plates of cases with severe prostration, a septic temperature curve and a bacterial report of streptococcus hemolyticus, the negative finding of undestroyed intercellular bony walls is a clue to the fact that we are dealing with the hemorrhagic type of mastoiditis. In other words, failure to find bone destruction is substantiation of the diagnosis of hemorrhagic mastoiditis and usually indicates surgical intervention.

In discussing the laboratory aids at our disposal in the diagnosis of the nonsuppurative otitic diseases, I am sorry that I will not be able

to speak as definitely as I have of their help in the suppurative otitis. Our main problem in this field is deafness and I feel that laboratory medicine will do more to help solve this problem than anything we may do in perfecting the testing of hearing. All our hearing tests have until now done nothing but confuse us. They have afforded us a classification of deafness which is largely erroneous and which, used as a basis for investigation, has misled us. Permit me to digress for a moment to make clear just what I mean. For example, a hearing test may yield results characteristic of a so-called catarrhal deafness and yet an acoustic neurofibroma is found to be the cause of the loss in hearing. A typical hearing test of a so-called otosclerosis is found to be due to syphilis of the central nervous system. These are actual occurrences from my practice and I could quote many others.

I have accordingly come to view deafness from a different standpoint,—whether or not it is progressive in character. Some patients in whom a loss of hearing is present will never get worse, whereas others show a gradual progression of the loss in hearing acuity. Consequently, the division of deafness into progressive and nonprogressive types permits a study of this condition from a new angle; namely, what causes the deafness to progress? That the cause may be different in each case is granted. Whether it is endocrine, toxic, infectious, the result of inherited latent characteristics or the product of faulty metabolism, is yet to be determined. In all probability each of these factors plays a role.

Blood Chemistry: In studying these cases, Almour and I have found that most patients with progressive deafness show a derangement of the calcium content of the blood. In cases of long standing a decrease in the calcium content is noted. In the more recent and active cases, an increase is more apt to be found. In conjunction with the calcium derangement, a great number of the cases show a high uric acid in the blood.

Just what the significance of this finding is I do not know. We originally reported it as an observation and as an aid in the diagnosis of chronic progressive deafness. While we have several theoretical explanations, we are as yet unable to definitely account for it. Nevertheless, I consider it a valuable diagnostic aid in helping to differentiate the progressive from the stationary types of deafness.

Wassermann: The Wassermann test should be a routine procedure in all cases of deafness. Syphilis does not always yield the typical acoustic reactions of a nerve deafness, either with the forks or with the audiometer. Where, on examination of the blood, a negative

Wassermann is obtained but syphilis is strongly suspected from the clinical data, a spinal fluid Wassermann and colloidal gold test are advisable.

X-ray: In the diagnosis of deafness, too, the X-ray holds forth a possibility of help. Joseph C. Beck, in 1915, made a preliminary report of the finding of changes in the long bones in cases of otosclerosis. With a view to checking his findings and learning what possible lesions might be presented, I had Dr. Henry K. Taylor make a Roentgenographic study of the long bones in several of my cases of deafness. In those cases wherein I had made a diagnosis of progressive deafness, Dr. Taylor reported changes in the periosteal coverings of some of the long bones. In two of the cases he found early evidences of cystic formation in the cranial vault indicative of an early Paget's disease. In those cases which were nonprogressive in character, no abnormalities were found in the skeleton. Here also I am unable to explain the findings. I simply submit them for your study and future investigation.

The correct estimation of laboratory criteria removes many cases in otologic practice from groups to which preconceived routine therapy is applied. It permits an individualization of treatment according to the given case because it furnishes data so significant as to differentiate conditions which appear clinically alike. Thus details of pathology are better comprehended, the management of cases becomes increasingly resourceful and results are more uniformly successful.

In conclusion, I desire to stress the importance of the role which bedside observation plays in all otological cases and to emphasize the need of accurate clinical observation. In the last analysis, it is good clinical judgment which furnishes the foundation upon which laboratory aids build in making diagnoses.

51 West 73rd Street.

THE NEW YORK ACADEMY OF MEDICINE.

SECTION OF LARYNGOLOGY AND RHINOLOGY.

Regular Meeting, Feb. 23, 1928.

(Continued from page 368.)

DR. A. P. VOISLAWSKY: What is your experience with postoperative trauma —pain—time of healing? Will you give us your method of postoperative treatment? (Another query about the lower pole?)

DR. L. T. LEWALD: I would like to know something about the postoperative complications, as to the possibility of aspiration or embolic abscess of the lung. Is there any more likelihood of freedom from that complication?

DR. HERMAN: First, allow me to thank you for your kindly discussion. Before answering any questions, please let me say that I shall be very glad to accept Dr. Smith's invitation, which I consider an honor.

To Dr. Fowler I will say nothing is easier of proof than the success or failure of the operation according to this standard. If the excised tonsil shows an intact capsule, then the ablation has been complete, and in the vast majority of cases such an intact capsule may be demonstrated.

In regard to Dr. Nilsen's statement that in his 30 or more cases done by this method he has used the large body electrode placed at a distant point, I wish to say that I do not use that technique any more, as one has no control whatsoever over the current penertation, which is directed into the tonsil fossa, thus at times causing too great a burning and subsequent sloughing. With the newer technique, that is the dispersing electrode being attached to the seizing forceps, the current is directed into the tissue which is being removed. Therefore, one is able to use almost any current strength. In fact, the tonsil upon removal can be found to be steaming, but upon looking at the fossa there will be but a very slight searing having taken place. That will also answer the question in regard to the large vein sometimes noticed in the fossa. The coagulation effect is so slight that I have never had any untoward results.

In answering Dr. Voislawsky's question in regard to hemophliacs will say that I am perhaps more afraid of them than ever. All of my work in the development of this instrument having been done in private practice (of course, not meaning to imply that just as much care and consideration would not have been given to clinic cases), it has been a most essential requisite that there should be no untoward results. It has thus taught me to take more than necessary care, when I have a longer than usual coagulation time to deal with.

As to postoperative sloughing, I do not think this occurs as often as in other methods, owing to the absolute sterile operating field (it having been made sterile by the searing effect).

In regard to the adaptation of this procedure to all tonsils, I do not believe there to be an absolute specific for anything in life. I do believe, however, that this instrument is adaptable—or I should say it has been in my experience suitable in about 98 per cent of the cases I have operated upon.

As to dissection: One may use the ordinary dissection method, as it is not in the primary dissection that the bleeding occurs but in the latter part. The loop being interchangeable with the open snare, as illustrated by one of the slides, it is possible to make the final step of the dissection with the electrified open snare.

About breaking the adhesions before engaging the tonsil, I find that in children and elderly people the tonsils are readily adaptable to the fenestrum of this instrument. It is in the middle-aged, in my experience, where the tonsils are tied down. As a rule, one is able to break up these adhesions by forcing them through the enucleator, thus making them quite adaptable to the electric tonsillotome.

In regard to Dr. Smith's trouble with the electric current, I do not know just what his experience was, as having once set the electrode for the proper deliverance of current, one is not obliged to pay any more attention to it. In so far as the manipulation of the current interfering with the technique of the operation, I will say that it does not do so in any way; the technique is identical with that of the Beck-Mueller technique. Excepting that after having engaged the tonsil through the fenestrum, and drawn the wire taut, the seizing forceps are closed as explained in the paper, thus making a thoroughly good electric connection, all one has to do is to place his foot upon the foot switch and draw the snare wire home.

Sparks and Ether: This is a question that is always asked. As stated in my paper, I have done many tonsils under general anesthesia, using ether. The possibility of a short circut caused by the electrodes coming in contact with each other, while in the throat is very remote, as in the event the snare wire would break it would immediately be drawn into the insulated tube. However, I have conducted experiments on animals anesthetized with ether, and subsequently placing the mouth gag and striking the two electrodes together within the recesses of the throat, causing a multiplicity of sparks, without any unfortunate results; nor have I been able to ascertain from any authority, to date, of anyone having had trouble. I am also aware that at various large clinics work on malignancy of the tongue, this type current is being used under ether.

Postoperative Trauma: I do not think that the Beck-Mueller technique is resultant of as much trauma as one finds after having used the dissection and snare method. As for the healing time, it is a trifle shorter. In that, the throat immediately after operation has the appearance of the usual day-old throat.

Postoperative Treatment: I do nothing excepting to advise aspirin dissolved on the tongue, at intervals, for pain. I forbid all acid fruits, particularly bananas and tomatoes.

Remaiinng Lower Pole: This happens only in those instances where there exists bands of adhesions due to prior peritonsillar abscesses. In this event, it is very easy to determine and all one is obliged to do is to pick it up with the insulated forceps and snare it off with the electrified open snare.

Aspiration: The aspiration of blood, etc., is reduced to an absolute minimum in this method, as its use keeps the throat clear of debris at all times.

Unusual Lesions of the Nose and Throat, Including Trichinosis of the Tonsils. Dr. L. H. Meeker.

(To appear in a subsequent issue of THE LARYNGOSCOPE.*)*

DISCUSSION.

DR. A. A. EGGSTON: I am quite sure that Dr. Meeker has convinced us that we do get a curious collection of tissues in examining specimens from the nose and throat. The examination of material coughed up is also quite important, and not infrequently it is quite possible to make a diagnosis of malignancy of the lung.

The examination of the tonsils to determine their etiological factor in a toxemia is the most unsatisfactory examination one has to make. It is very difficult to say that a tonsil is undergoing hyperplasia, and frequently very difficult to identify an abscess as one that is absolutely embedded. The presence of cysts is not uncommon, probably resulting from adhesive processes in the crypts and inclusion of epithelium with the accumulation of debris. I wonder how much pathological importance can be attached to these formations?

As for saying that tonsils are infected histologically, I don't think anyone has any right to make any positive diagnosis. The physiology of the patient is the last criterion as to whether a tonsil is the source of a toxemia.

The examination of the tissue from the nose has been of much interest to me from the point of explaining the processes that go on in sinusitis. Undoubtedly, as a result of repeated infections, such as acute colds, the mucous membrane of the antrum and other sinuses of the nose will show permanent chronic histological changes, that ultimately affect the functions of the mucous membrane and the health of the individual. Frequently material from the ethmoid and sphenoid sinuses show distinct pathological changes in the blood vessels and the character of the membrane will depend on the character and

type of the pathology. I have been particudarly interested in noticing in the polypoid type the process of thickening and occlusion of the veins and lymphatics obstructing drainage and a polypoid condition results. In other cases showing atrophy of membrane and bone, the vascular changes are chiefly arterial, with more or less diminished blood supply, and therefore atrophic changes occur in the parts.

There is frequently found a combination of the two processes where you have thickening and absorption of the bone, as well as polypoid formation. I believe the examination of the tissues in the sinusitis will some day help to explain the changes that take place in the nose and throat following repeated attacks of acute colds. I have not yet clearly classified the findings in my mind, but there are underlying conditions that bring about atrophy of the mucous membrane, absorption of bone and polypoid conditions.

Where there are distinct eye ground changes, with optic nerve involvement, the examined material from the ethmoid and sphenoid frequently show very little if any pathology. It is a question of quantity of cellular reaction in the mucous membrane of the nose. There is in the mucous membrane, a great amount of cellular infiltration normally and it is frequently difficult to evaluate the amount of reaction shown in a section.

In reference to the trichinae and other animal parasites, I was much impressed some years ago in studying a cyst taken from the conjunctiva in which was found a beautiful example of echinococcus cyst. The patient was carefully examined physically and by X-ray, and the case has been followed up carefully to see if there was any evidence of similar cysts in other parts of the body, but nothing of the kind was found; it was apparently a direct infection of the mucous membrane of the eye by the echinococcus hooklets.

We have had several specimens of adherant thyroids removed from the base of the tongue and tonsil region. After removal these patients have all suffered from lack of thyroid and had to be given thyroid extract.

The presence of fungi in tonsillar crypts has received a great deal of attention; however, the exact significance and classification of these organisms has not been determined. Some of them certainly appear like some types of actinomyces.

DR. MEEKER: Dr. Warthin reported our case of actinomycosis in human tonsils but later it was recognized that true actinomycosis in human tonsils has not been proved to exist. The similar colonies seen in the tonsils are quite different members of the same family of molds.

Lymphosarcoma of Tonsil. Dr. John M. Lore.

This patient, B. N., male, age 41 years, was first seen by me Oct. 8, 1925. About three weeks before this he developed a sore throat, which was diagnosed and treated for diphtheria, but with no improvement in symptoms. When seen by me he still complained of soreness in his throat, especially on swallowing, and also of hoarseness of two days' duration; recently developed a cough.

Past history revealed a penile sore when a young man. He received treatment for it. Examination of the throat showed a very large right tonsil, which seemed to encroach considerably on the soft palate above and which extended below into the pyriform fossa. There was a deep ulcerated area located about the middle third of the mass. It was hard, fairly well fixed to the surrounding tissues, did not present an appearance of an acutely inflamed mass, nor did the ulcerated area bleed. The left tonsil looked normal. The larynx showed both cords to be congested. There was some crusting present. There were a few small glands palpable on the right side of his neck, both axilla and inguinal regions.

Clinical workup: Blood Wassermann negative. Smears from ulcer negative for Vincent's angina.

Operation Performed by Dr. Lore, Oct. 14, 1925: Examination of right tonsil showed a large mass, apparently enlargement of the tonsil itself. This mass extended into the right pyriform fossa. It was hard to the touch, and in the region of the upper pole there was a deep sulcus. This mass was removed in toto by making an incision through the anterior pillar down to the capsule and dissecting it from its bed with sharp dissection. A frozen section showed that the tissue was a lymphosarcoma. There was no unusual bleeding and this was easily controlled. The left tonsil was not touched.

Dr. Wadham's Note, Oct. 15, 1925: Cervical, axillary and inguinal groups of noles moderately enlarged. Painless, movable without periadenitis. Pathologist's report on tonsil confirms diagnosis of lymphosarcoma. Radiation could be tried, but disease is general rather than localized to one group, so that only temporary result would be obtained. Suggest X-ray of chest and X-ray of neck and tonsil if final report is sarcoma. If throat improved definitely under medication, the other groups could be radiated.

X-ray Report, Oct. 17: No definite radiographic evidence of pulmonary metastasis. Pulmonary fibrosis moderate and symmetrically distributed.

Blood Examination, Oct. 17: R. B. C., 5,500,500; hemgb., 90 per cent; W. B. C., 9,600; poly., 65 per cent; lymph., 33 per cent; M. and T., 0; spec., baso. 2 per cent.

Urine Analysis, Oct. 14: Negative. Char., amber; react., acid; Sp. Gr., 1,018; alb., 0; sugar, 0. Special microscopical: Cast one hyaline and finely gran., leu +, Epith. Flat X.

P. O., Oct. 15: Char., amber; react., acid; Sp. Gr., 1,020; alb., 0; sugar, acetone.

Pathologist's Report: Specimen consists of multiple masses of yellowish-white tissue, the largest about the size of a hazel nut, showing rough, fragmented surfaces with the presence here and there of small tags of brown tissue having a fasciculer appearance and suggesting striated muscle grossly. On section the masses cut with little resistance, revealing a yellowish-white homogeneous surface.

In the histological sections the loss of the normal architecture of the tonsil is manifest. Only in one or two places can the lymphoid follicles be recognized, their structure being poorly preserved, both they and the normal lymphoid tissue of the organ being replaced by a diffuse outgrowth of cells having large, round, pale nuclei that take the basic stain poorly. For the most part there is a regularity in the size, shape and staining characteristic of these cells, although they are placed where they are anoplastic and exhibit atypical mitosis. They lie in close arrangement for the most part also and show the presence of thin strands of an acidophilic hyaline material that tend to divide them into irregular lobules. The sections examined do not show the presence of any limiting fibrous capsule to the tonsil, but rather is there evidence of cellular infiltration of the striated muscle. These cells lie in the intrafascicular connective tissue and in many places the infiltration is so dense as to present the picture of isolated muscle fasciculi marooned in dense cellular outgrowth. The tumor is relatively scant in blood vessels.

The picture is that of a lymphosarcoma of the tonsil, probably of the reticulum cell type, and shows locally invasive characteristics.

In view of the tissue report, X-ray therapy was advised and carried out by Dr. Maver. Following the last X-ray treatment he had a severe laryngeal reaction, from which the larynx has not fully recovered. Up to date there have not been any signs of recurrence or metastasis.

Teratoma of Nasopharynx. Dr. Louis Lawlor.

About 1924, L. L., was presented for diagnosis before this Section. Since then the diagnosis has been established and the condition cured.

Briefly, the history was that about 24 years ago he noticed for the first time a small swelling in the left side of his throat. Not causing him any discomfort, he did not do anything about it. The growth, however, showed a gradual increase in size, so much so that at the time he presented himself at Dr. McCullogh's Clinic, Manhattan Eye, Ear and Throat Hospital, he complained of difficulty in breathing, speech and eating solids.

Examination at that time revealed an enormous mass, entirely filling the nasopharynx, oropharynx and extending down to the larynx, and anteriorly as far forward as the hard palate. The uvula was seen at the right side of the throat. The mass was smooth and rather firm. Some glands were palpable on the left side of his neck. Medical examination shows nothing of interest.

Blood Wasserman, 4+. X-ray of neck, Jan. 5, 1924: There is considerable swelling in posterior pharyngeal wall, but unable to differentiate the condition. The entire pharynx seems to be filled. X-ray, July 10: After diagnostic puncture and injecting bismuth into mass, there seems to be a mass filling the pharynx, extending to the epiglottis. The bismuth collects in a pocket in front

of the epiglottis. After a course of antisyphilitic treatment the Wassermann became negative and is still negative.

Blood chemistry and urinalysis were negative. Radium treatment, April, 1924, external block used had no effect on mass.

In May, 1924, an emergency tracheotomy was performed and June 6 was discharged with thracheotomy tube in situ.

Patient was readmitted to the Manhattan Eye, Ear and Throat Hospital on July 22, 1924, and operated upon July 26. Anesthesia by Dr. Flagg. Ether used through a catheter inserted into trachea through the tracheotomy tube. It was considered safest to have the external carotid artery (left side) under complete control. Therefore, an incision was made along the anterior border of the sternocleidomastoid muscle and by proper dissection the external carotid artery was exposed and recognized. A small tape ligature was placed around it, but was not tied. In approaching the artery, the glands which were palpable were located and removed. Attention was then transferred to the pharyngeal growth itself. A side mouth gag was used to keep the mouth open. An effort was made to place a gauze packing below the growth to prevent blood and other secretions from getting into the larynx, but with little success.

The anterior pillar of the left tonsil was barely made out and using this as a landmark an incision was made at this point, extending from above, down parallel to the pillar. The plane of cleavage of the tumor mass was easily found and this was followed by the finger and blunt scissors dissection until the mass, which now felt cystic, was completely dissected except for the pedicle of attachment, which seemed to be under the surface of the left sphenoid bone.

Inasmuch as the mass was too large to deliver through the mouth in one piece, it became necessary to remove the lower two-thirds first, ligating first the upper stump. The remaining portion was removed by means of finger dissection. The cavity created by the removal of the cyst was now swabbed with solution mercurochrome 2 per cent. The incision in the soft palate was partially closed by means of interrupted silk sutures and cigarette drains were used. The mouth was then lightly packed to prevent the cavity from filling up with blood.

The bleeding during the entire operation was less than the amount one gets in the average tonsillectomy.

Two days later, the ligature placed around (but not tied) the external carotid artery was removed. He had an uneventful convalescence. The mouth packing was removed 24 hours later. The wound was clean; all drains were removed in four days. On Aug. 8, 1924, he was discharged to return to the clinic for treatments. On discharge there was quite a bit of redundant tissue still present, but gradually disappearing. The neck wound was healed—tracheotomy tube still being used, general condition good.

As soon as the mouth wound was all healed, the tracheotomy tube was removed. He is now all healed, with practically no scars.

July 26, 1924: The specimen consists of a very irregular macerated and torn mass of tissue 8x7x6 c.m. The tissue is apparently fragments of a cyst wall, the inner surface of the cyst is covered with caseous material and necrotic tissue. In other areas the tissue is very dense and upon sections contains dense fibrous tissue, cartilage, areas of necrosis and hemorrhage.

Microscopically, there is a dense stroma of hyaloid connective tissue and in areas of cartilaginous tissue. Scattered throughout the stroma there are irregular grandular structures, some of which are cystic. There is a marked proliferation of the epithelium in the glands. In areas the epithelial cells form irregular nests of cells without definite limiting membranes. A few of the cells show mitosis. The stroma shows a dense infiltration of mononuclear cells and polymorphs.

Diag. mixed tumor (teratoma). In areas there appears to be carcinomatous degeneration. However, these mixed tumors are of slow growth, they do tend to become malignant and are likely to recur.

July 26: The specimen consists of a firm nodular mass of tissue, 12x8x 10 m.m. Microscopical sections show an irregular conglomeration of mucous glandular structure. The epithelial cells of the glands are hyperplastic and some are secreting mucus. There is a slight stroma between the glands.

Diag. mixed glandular tumor, a part of the teratoma.

Diag. hyperplastic lymphnode with suspicious metastatic tumor areas.

I wish to take this opportunity of expressing my gratitude to Dr. Samuel McCullogh and Dr. Francis W. White for their kind advice and assistance.

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY.

March 20, 1928.

Proliferating Retinitis. Dr. Harold L. Goss.

J. A. P., male, age 26 years, a bookkeeper, was first seen by me in November, 1927. Since July, 1927, he had been going to different doctors for blurring in left eye. The extraocular examination was essentially negative. Ophthalmoscopic, both eyes show large floating vitreous opacities, with a proliferative retinitis and new-formed blood vessels. In both eyes there were several areas of recent hemorrhage throughout the vitreous. Foci abscessed teeth removed, infected tonsils removed, negative for tuberculosis, immediate treatment was sweats and catharsis. He has had no hemorrhage since removal of foci.

Vernal Conjunctivitis; Two Cases. Dr. H. A. Schoffman.

Dr. Schoffman presented two boys, not related, both age 11 years, born and raised in Seattle. The pathological manifestation in one is of the conjunctivitis of the upper lids, in the other of the limbuses only.

One has had it for seven years, the other for five years. History and physical examination negative. Cutaneous tests for protein sensitivity negative, no foci of infection determinable. Last summer, 1927, they both had radium treatments consisting of 50 m.gr. in 1 m.m. brass capsule, rubber-covered, and held ½-inch from the anterior surface of the eyeball. One-hour treatment each week for five weeks. This made a total of 250 m.gr. hours of gomma radium rays to each eye at a distance of ½-inch within one month.

As the use of radium on the eyeball is quite new and accompanied with danger, the dose and quality of rays was decided upon after careful consideration of the recent literature of experimental work in this line, and the eyes were carefully watched during the treatment. The treatments were given by Dr. H. B. Thompson. At the present time the manifestations are returning.

In discussing Dr. Schoffman's cases, Dr. Goss was of the opinion that the amount of radium used and the length of time employed, as described by Dr. Schoffman, was too small to be of benefit. He had seen several cases in the South apparently cured with much larger doses and longer exposures, using plaques beneath the lids.

Dr. McCoy has seen benefit follow excising the papules and touching the base with stick silver nitrate.

Glaucoma in a Case of Acromegaly. Dr. W. G. Cameron.

Dr. Cameron presented the case of J. L. H., switchman, age 56 years. I am presenting this patient because, as you can see, he is a typical case of acromegaly, as well as for his eye condition. I first saw him this afternoon and so cannot give any laboratory findings or much information concerning his general condition.

His left eye had broad iridectomy by Dr. Cooke in 1918, at which time he stated that the right eye had good vision. I cannot get any definite history of the visual acuity of right eye except that the patient says that he had no pain and so did not pay much attention to it so long as the vision in the left eye was good. Vision in the left eye has been good until the past few months and now he does not see as well as before. Vision, right eye, 6/60; left eye 6/12; tension, 42° each eye.

The right eye has active pupil and clear media. Large excavated glaucomatous cup. The left eye shows coloboma of a very nicely done iridectomy and I believe the resulting 6/12 vision after 10 years is very good.

There is a question how long vision in right eye has been reduced as we now find it, and if destruction in optic nerve is such that operation will be of no benefit. He will go into the hospital for thorough examination of general condition and visual fields and it will be of interest to find out the condition of the pituitary body and see if that is causing atrophy as well as this glaucoma.

Skin Graft in Eye Socket. Dr. L. L. McCoy.

C. D., age 19 years, a state industrial case, was seen July 31, 1926, with a very severe lime burn of the left eye. The cornea was paper-white. Under

hospitalization and treatment the cornea cleared up very markedly and adhesions were kept down for a long time. Finally a symblepharon started, which became complete in about a month. The cornea began to get hazy again and in about six weeks there seemed to be a complete leukoma. In three or four months glaucoma developed and enucleation became necessary. This healed up well, but the socket was so contracted that wearing a prosthesis was impossible. On May 3, 1927, restoration of the orbit by means of the Wheeler operation was done and a very satisfactory result followed. During the past few months mucus secretion has been growing until at the present time it is very profuse and annoying.

This case was presented for the purpose of getting some idea regarding the allaying of the secretion.

Keratoconus. Dr. L. L. McCoy.

Miss S. S., age 37 years, was first seen Nov. 12, 1927. She has a marked keratoconus; left worse than right. Vision, right 20/200; left, practically nil. Glasses improved the right vision to 20/30, but no improvement on the left. Interior of her globes were practically normal.

Persistent Hyaloid Artery. Dr. L. L. McCoy.

M. H., school girl, age 12 years, has the complete remains of a hyaloid artery, extending from the posterior surface of the lens to the center of the disc. There was a slight point on the posterior surface of the right lens, also the remains of a hyaloid artery. Vision in the right eye was normal, in the left 20/40 with glasses. Eyes were otherwise normal.

Congenital Nuclear Cataracts. Dr. L. L. McCoy.

D. B., school girl, presented a case of small congenital nuclear cataracts. When the pupils were contracted vision was quite reduced, but when semi-dilated was fair. Eyes otherwise normal.

In discussing the cause of the profuse discharge from the eye socket following skin grafting, Dr. Davies said he thought the discharge was probably due to too thick a graft, and advised removing the graft and replacing with one cut very thin, continuing only the upper layers and avoiding sweat glands.

Bronchoscopic Diagnosis of Aortic Aneurism. Rupture Into Bronchus. Case Report. Dr. J. A. Weber.

Patient admitted on medical service of hospital on June 30, 1927. Male, age 45 years, outdoor laborer. Complaint: Intermittent attacks of tightness in chest and dyspnea for past two winters. General malaise and weakness of six months' duration and especially during the past two months. Slight productive cough.

Physcal examination unimportant except for dullness, diminished breath sounds, and increased fremitus over the base of the left lung. Heart negative. Blood pressure, 100/65. Temperature, 99½°. Pulse, 76. Blood Wassermann, 4 plus. Urine, negative.

Fluoroscopic examination showed slightly broadened aortic arch. X-ray same. with some evidence of lung suppuration in the left base. A provisional diagnosis of bronchial stenosis was made and a diagnostic bronchoscopy ordered to determine the presence or absence of the bronchial stenosis, and to determine what caused the lung pathology. Ordinarily we do not bronchoscope luetic cases, but in this case we knew he had syphilis and we also knew that he had pathology we had not accounted for.

Diagnostic bronchoscopy was done on Aug. 2, 1927, showing a very markedly pulsating aorta and a compression stenosis of the left main bronchus, about 1 inch from the bifurcation. There was no ingress or egress of air through the constriction. The bronchoscope, 7 m.m., was passed through and considerable mucopurulent secretion aspirated. Negative for tuberculosis, few pus cells and many bacteria. Aug. 12, 1927, bronchoscoped again, but I did not go through the constriction. Findings were the same. Thirty c.c. of lipiodol were injected and X-ray showed it to have all run into the right lung.

The patient improved on antiluetic treatment up to March 6, 1928, when he died suddenly while walking on the street.

This case was presented to show the value of diagnostic bronchoscopy and because of the scarcity of literature on the bronchoscopic findings in aortic aneurism.

A. E. Hillis, Sec.

NASHVILLE ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY.

February 20, 1928.

Two Cases of Mastoid Empyema Associated With Empyema of the Maxillary Sinus On the Same Side. Dr. M. M. Cullom.

James C., white, male, age 18 years, counsulted me on Feb. 6 for discharging ear. He gave a history of a discharging ear at intervals for more than a year. Pus was seen issuing from a perforation in the left drum membrane, accompanied by pulsation. The patient denied any pain. There was absolutely no tenderness over the mastoid. Temperature normal. The only thing the patient noted was a throbbing sensation in the ear. The boy was attending to his duties in school and taking part in all its activities, social, athletic and otherwise. As the patient was in school in a town some 50 miles away and was only in for the day, I gave him a saturated solution of borasic acid and alcohol for instillation, with strict instructions to come back the following week. In my notes I made the entry, "probable empyema of left mastoid". He returned in a week with absolutely no change in appearance and symptoms. I had an X-ray picture made of the mastoid and accessory sinuses. As you see, the mastoid is quite large, with cloudiness and evidence of breaking down of the cells. The X-ray of the accessory sinuses showed a definite cloudiness of the left maxillary sinus. This is in line with my observation for the past seven years. At least 85 of my simple mastoids have shown an infected antrum on the same side as the mastoid. The patient was operated on Feb. 14 at St. Thomas Hospital. The entire mastoid was involved. There was a perforation of the inner plate.

Mrs. C. C., age 25 years, consulted me July 2, 1926. She had a well developed empyema of the left mastoid. X-ray of the mastoid confirmed the diagnosis. An X-ray of the accessory sinuses showed the left antrum cloudy. She was operated on at Vanderbilt Hospital July 8 and had an uneventful recovery. She declined to have the antrum drained. The interesting point is that on Jan. 24, 1928, she returned with an acute empyema of the left mastoid, which necessitated a reoperation. The question is, did the infected antrum bring on the second attack?

Discussion: Dr. Robert Sullivan said that he thought that Dr. Cullom's observation of empyema of sinuses and mastoid was interesting. He hadn't had many cases, but recalled three cases where this observation was true. No doubt it is often true if we look for it.

Dr. E. B. Cayce said that several years ago a report came from Scotland of some man who reported out of four or five hundred postmortems that 68 per cent of the antrums, taken irrespective of cause of death, were infected. At that rate it looks as if there would be at least four infected antrums in the meeting tonight.

Dr. Fred Hasty said that he and Dr. Cullom had reviewed their mastoid cases and had found that a large percentage had had paranasal sinus infection. Most of the patients were operated on for one of these troubles and we are not conscious of the other condition. Dr. Hasty asked Dr. Cullom if the boy had any pain.

Dr. M. M. Cullom replied that there had been discharge at intervals, but no pain.

Dr. Fred Hasty then asked if there was any history of tuberculosis.

Dr. M. M. Cullom replied that the family history was very good, and that the patient was a good specimen.

Dr. Fred Hasty then explained that he thought mastoiditis without pain brought up the question of tuberculosis. He thought that practically all of such cases were considered tuberculosis or diabetes. While we are considering

this subject I think the possibility of there being a marked difference in the bacteria of the organism that will grow and develop in a paranasal sinus and mastoid should be thought of, for the mastoid is one of the paranasal sinuses. It is connected by longer tube.

Dr. E. L. Roberts asked if he understood Dr. Hasty to say that mastoid abscesses not accompanied by pain were to be suspected of tuberculosis.

Dr. Fred Hasty replied that he thought so.

Dr. E. L. Roberts said that he recalled a child, age 6 years, who developed what appeared to be a very acute mastoiditis. The child was in good condition, running around, and had a good appetite. The severe swelling went down after a few days and the condition looked almost normal and the child did not complain of any pain or discomfort. Pressure over the mastoid region revealed fluctuation and he couldn't understand why she had had so few symptoms, and the condition really seemed to be subsiding. I had suggested operation, and finally the family consented. I made the incision through the soft part and pus oozed out and was all in the soft parts. There was a good size hole in the inner plate. I never had given a thought to T. B. in this case until Dr. Hasty mentioned the absence of pain, and the swelling, which had been extreme, subsided. Where did the pus go to? The drainage in the middle ear had stopped.

Dr. Cullom, in closing, said that he rarely ever saw a middle ear abscess in connection with an infected antrum that will not go on to mastoiditis in time if it is not corrected. It seems that the infection from the antrum to the mastoid promotes this. Of course, the drainage can go down through the mouth, Eustachian tube, or by blowing the nose, and drive the pus up into the ear. Dr. Hasty and I went over these cases and felt that a majority of the cases of mastoiditis in adults were due to sinuses and that these were all practically always on the same side with the mastoid.

Herschel Ezell, Secretary-Treasurer.

February 20, 1928.

Exostosis of the External Auditory Canal. Dr. E. B. Cayce.

R. W., male, white, age 21 years, referred by Dr. R. E. Warren, of Pulaski, Tennessee, and Dr. S. T. Ross, of Nashville, gave the following history: Had diphtheria, scarlatina, whooping cough and measles when a child. Has had what was diagnosed as chronic appendicitis since he was age 15 years. Appendix was removed two years ago.

Nose history: When he was age 5 years he had a severe fall on the nose. About three years ago the nose was broken in a football game. On examination the nasal septum was pushed to the right side, and it was impossible for him to get any breath through that side except by an unusual inspiratory effort. The inferior turbinate on the left side was hypertrophied.

Ear history: He complained of a pain in the ear when exposed to a strong wind two years ago. At that time he was driving a truck, and first noticed early in the morning when the wind struck his ear that there was some discomfort. Later on his hearing in that ear became defective, and he compained of a feeling of the ear being stopped up.

On examination of the ear it was impossible to get even the smallest baby-sized speculum in so that the eardrum could be examined. The X-rays which were exhibited showed as follows: Frontal sinuses very large and clear. Right antrum slightly cloudy and on the floor of the antrum there is a shadow of a good size tumor mass, which resembles a polyp. Sphenoid area clear and sella turcica normal. Left mastoid large, pneumatic in type, cells are all clear and extend well anterior to the canal. Right mastoid large, pneumatic in type and cells extend well anterior to the canal, and cells are slightly cloudy. This looks like an acute mastoid.

I advised a submucous resection of the septum and hoped to be able at the time to get enough cartilage to slip up under the skin over the depression in the bridge. Owing to the fact that it was a traumatic fracture we were not able to get enough cartilage to fill in the depression.

After doing a submucous resection a few whiffs of gas were given, and with a curet the specimen shown was removed. As you will see, it was only attached in about a third of its area, and came out very easily. The exostosis gave the X-ray of the mastoid the appearance of broken down cells.

Dr. E. B. Cayce presented his patient.

Discussion: Dr. Herschel Ezell asked if he understood that the tumor was removed through the external auditory canal en mass.

Dr. E. B. Cayce replied in the affirmative, showing Dr. Ezell where he put the curet.

Dr. Robert Sullivan then asked if the ear operation was done under local.

Dr. E. B. Cayce said that he did the submucous under local, and then Dr. Ross gave gas for the removal of the mass, but he said that had he known how slight the attachment was he would not have used gas at all.

Dr. Herschel Ezell asked if there had been any discharge from the ear.

Dr. E. B. Cayce said that there had been no history of any discharge.

Dr. Herschel Ezell said that he was glad to have seen this case. He had only one patient like it and the bony-like growth was not as large as in Dr. Cayce's case. He noticed that in this case, and it was true in his case, that the growth was located at the usual point, that is, at the junction of the membranous of the osseous canal. However, they are not always located there. I noted in reading up on this subject that Ballanger makes a difference between exostosis and hyperostosis. However, some men consider it one and the same thing. The difference is that hyperostosis is the general enlargement of the area and exostosis is a more or less pedunculated and accessible bony growth. It is interesting, though, that the case was not associated with middle ear trouble.

Dr. E. B. Cayce in reply said that the drum did not show any scar.

Dr. Herschel Ezell asked if a blood Wassermann was made to see if it was due to lues.

Dr. Fred Hasty said that this type of case was very simple to deal with. The more simple the less complications. I think the technique Dr. Cayce employed in this case, that is, simply slip your curet around until you feel it and gradually bring it out, is best. The care is due to the fact that X-ray shows that something is abnormal about the bone and it is possible that the facial nerve may not be in its usual location. All cutting should be done in line with the facial nerve.

Dr. M. M. Cullom said that he had never seen a case of exostosis outside of the hospital, that is, he had never had one in his private practice.

Dr. E. L. Roberts asked why Dr. Ezell might suspect syphilis as a cause for this.

Dr. Herschel Ezell replied that syphilis was given as a cause by the text-books. Also gout, hereditary, etc.

Alfred E. Smith, Governor of the State of New York, has designated Dr. Otto Glogau, of 1130 Park avenue, as Delegate to represent the State of New York, at the Fifth International Medical Congress on Occupational Diseases, Budapest, Sept. 2-8, 1928. Dr. Glogau will read a paper on "Occupational Diseases of the Ear, Nose and Throat Among American Factory Workers", the results of oto-laryngological examinations made by him for the New York Factory Investigation Commission, published in its official report.

THE PHILADELPHIA LARYNGOLOGICAL SOCIETY.

COLLEGE OF PHYSICIANS.

Meeting of April 24, 1928.

The Technique for Operating Intrinsic Cancer of the Larynx by Laryngofissure. Sir St. Clair Thomson.

DISCUSSION.

DR. RALPH BUTLER: I would like to ask Sir St. Clair Thomson's opinion about taking a piece for examination before operation and also his advice in regard to using radium in operable and inoperable cases.

DR. F. O. LEWIS: I should like to ask if diathermy has been used at any time for laryngofissure.

DR. ROBERT RIDPATH asked if Sir St. Clair Thomson removed the aryepiglottic fold on the side on which the disease is present.

SIR ST. CLAIR THOMSON said removing a piece of tissue for examination in suspected cases has its disadvantages. If the removed piece is positive for malignant growth, one can go ahead at full speed and be radical in the treatment, but if the result is negative, then the situation is different. Valuable time is lost and there is a possibility that stimulation of the growth may occur. It must also be remembered that when the growth is deeply imbedded an adequate piece for examination is often very difficult to obtain. In more than half of the 70 cases that he reported, Sir St. Clair Thomson made the diagnosis of malignancy from the simple clinical examination alone. Syphilis and tuberculosis are sometimes difficult to differentiate from a malignant growth, but a Wassermann test, the employment of the X-ray and repeated sputum examinations usually lead to the proper diagnosis.

He said he never used radium except in hopeless cases or in people who read medical literature and insisted on radium therapy.

With reference to diathermy, he would always keep an open mind concerning it, although he could not see how diathermy could improve on laryngofissure or laryngectomy.

COMMUNICATIONS.

Editor of THE LARYNGOSCOPE, New York, May 31, 1928.
St. Louis, Mo.

Dear Sir:

In my article entitled, "Constitutional Deafness", published in THE LARYNGOSCOPE of May, 1928, I find that just below Figures 4, 5 and 6 there is a descriptive note under each which includes the words, "Author's observation". If you refer to my original article, you will find written at the lower portion of the page on which the photographs were attached that the pictures were taken from an article by Denjiro Nabeya, and I expected that the notations under each picture would indicate that they came from this article and were not observations of mine. This is extremely misleading to the casual reader and gives the impression that it was my intention to appropriate this work, when it is distinctly stated in the text on page 311 that the pictures were culled from the article by Nabeya.

I believe that a printed apology should be made in your next issue which would disabuse the minds of readers that there was no thought in my mind of committing plagiarism.

Yours very truly,

DR. MARK J. GOTTLIEB.